Registered at the G.P.O., Sydney, for Transmission by Post as a Newspaper. Published Weekly. Price 1s.

OCT 2 0 1927

THE MEDICAL JOURNAL OF AUSTRALIA

Medical Library

VOL. II.—14TH YEAR. SYDNEY: SATURDAY, SEPTEMBER 17, 1927. No. 12.

The 'Allenburys' DIET

A READY solution of the problem of feeding in most acute and chronic diseases, as well as in convalescence, is found in the use of "Allenburys" Diet. It is a complete, well balanced food, made from fresh full-cream milk and whole wheat, both of which are partially predigested during manufacture. This product is particularly valuable for invalids, convalescents, nursing mothers, dyspeptics and the aged. It promotes digestive ease and is well tolerated even in cases of extreme weakness. The "Allenburys" Diet keeps well, is readily prepared for use, and is pleasant to take.

The "Allenburys" Diet is quite distinct from the "Allenburys" Foods for infants.

Approximate Percentage COMPOSITION.

Milk Fat	15.4
Milk and Wheat Proteins	15.2
Carbohydrates (Lactose, Dextrin-Maltose, etc.)	62.9
Mineral Matter	4.3
Moisture	2.2

Calorific Value: 132.3 calories per oz. weight of powder.

A full sized package of this product will gladly be sent gratis to any Physician on request.

ALLEN & HANBURYS (AUSTRALASIA) LTD

13 MARKET STREET SYDNEY, N.S.W.

West of England All Wool Worsteds

Sac Suit to Measure

£9/9/-

New, original, each a subtle harmony of colour—there are many novel ideas in weaves and shades in these exclusive suitings just arrived at Peapes'.

The superior quality is unquestionable, the skilful interweaving of colours creating most attractive though reserved effects of Fawn, Brown, Grey or Blue.

Plain weaves in the same colours are also available.

In conjunction with Peapes' fine tailoring these suitings represent most unusual value at this moderate price £9/9/-

Patterns will be forwarded to any address promptly on request.

PEAPES

—for men and boys.
GEORGE STREET
(opp. Hunter Street)
SYDNEY.

THE MEDICAL JOURNAL OF AUSTRALIA

VOL. II.—14TH YEAR. SYDNEY: SATURDAY, SEPTEMBER 17, 1927. No. 12.

Authors of articles submitted for publication are requested to read the following instructions and to comply with them.

All articles must be typed with double or treble spacing. Carbon copies should not be sent. Abbreviations should be avoided, especially those of a technical character at times employed in ward notes. Words and sentences should not be underlined or typed in capitals. The selection of the correct type is undertaken by the Editors. When illustrations are required, good photographic prints on glossy gaslight papers should be submitted. Each print should be enclosed in a sheet of paper. On this sheet of paper the number of the figure and the legend to appear below the print should be typed or legibly written. On no account should any mark be made on the back of the photographic print. If no good print is available, negatives may be submitted. Line drawings, graphs, charts and the like should be drawn on thick, white paper in India ink by a person accustomed to draw for reproduction. The drawings should be large and boldly executed and all figures, lettering and symbols should be of sufficient strength and size to remain clear after reduction. Skiagrams can be reproduced satisfactorily only if good prints or negatives are available. The reproduction of all illustrations but especially of skiagrams entails the sacrifice of time and energy and is expensive. Authors are expected to take a corresponding amount of trouble on the preparation of their illustrations, whether skiagrams, photographs, wash drawings or line drawings. The references to articles and books quoted must be accurate and should be compiled according to the following scheme. The order should correspond to the order of appearance in the article. The initials and surnames of the authors, the full title of the article or book, the full (unabbreviated) title of the journal in which the article appears, the date of the issue (day, month and year) and the number of the first page should be given in this sequence.

Table of Contents

[The Whole of the Literary Matter in THE MEDICAL JOURNAL OF AUSTRALIA is Copyright.]

ORIGINAL ARTICLES— PAGE.

"Pyelitis in Childhood," by STEWART FERGUSON, M.D. .. 388
"Pyelitis in the Adult," by BALCOMBE QUICK, D.S.O., M.B., F.R.C.S. .. 389
"Puerperal Pyelitis," by ROBERT FOWLER, M.D., F.R.C.S. .. 393
"The Calcium and Phosphorus Content of the Blood in Fractures," by G. V. RUDD, M.Sc., A.A.C.I. .. 398
"A General Expression of Opinion, but not Necessarily an Expression of General Opinion," by R. W. HORNABROOK, M.B., Ch.B., M.R.C.S., L.R.C.P. .. 401

REPORTS OF CASES—

"Acute Dilatation of the Stomach," by ALAN E. LEE, M.D., F.R.C.S. .. 405

REVIEWS—

Obstetrical Difficulties .. 406

LEADING ARTICLES—

Our Universities .. 407

CURRENT COMMENT—

Empyema .. 408
Pyelitis .. 409

ABSTRACTS FROM CURRENT MEDICAL LITERATURE—

Gynæcology .. 410
Obstetrics .. 411

BRITISH MEDICAL ASSOCIATION NEWS— PAGE.

Scientific .. 412

MEDICAL SOCIETIES—

The Melbourne Hospital Clinical Society .. 417
The Newcastle Hospital Clinical Society .. 419

OBITUARY—

Andrew John Brady .. 421
George McIntyre Maggs .. 423

HOSPITALS—

The Cancer Ward of the Bendigo Benevolent Asylum .. 423

UNIVERSITY INTELLIGENCE—

The University of Sydney .. 423

PROCEEDINGS OF THE AUSTRALIAN MEDICAL BOARDS—

Victoria .. 423

BOOKS RECEIVED .. 424

DIARY FOR THE MONTH .. 424

MEDICAL APPOINTMENTS .. 424

MEDICAL APPOINTMENTS VACANT, ETC. .. 424

MEDICAL APPOINTMENTS: IMPORTANT NOTICE 424

EDITORIAL NOTICES .. 424

PYELITIS IN CHILDHOOD.[1]

By STEWART FERGUSON, M.D.,
Physician to In-Patients, Children's Hospital, Melbourne.

IN speaking on the subject of pyelitis in infancy and childhood, I regret I must say at the outset that I can bring forward nothing that is new nor state any original views on the matter. I wrote an article on this subject for THE MEDICAL JOURNAL OF AUSTRALIA in 1914 and on rereading it I have found my own views practically unaltered, nor does a perusal of the literature bring forward anything very different. The lesion is due to an infection of the pelvis of the kidney with the colon or coliform type of organism. The mode of infection has been the subject of much discussion as to whether it is an infection through the lymphatics or blood stream or simply an ascending infection through the urethra upwards. Its frequency in the female and especially in infancy when vaginal soiling with fæces can so readily occur lends favour to the latter route. However I intend to limit my remarks to the clinical rather than the academic aspect.

Symptoms.

As regards symptoms the usual history, especially in infancy, is one of some bowel disturbance with a tendency to diarrhœa, then a varying number of days afterwards the child becomes febrile. There are no localizing symptoms or anything suggestive of pyelitis. The degree of toxæmia varies considerably; sometimes the patients are very ill and toxic, at other times not more than they would be with an ordinary rise of temperature.

Some patients manifest definite signs of meningeal irritation. The child resents any interference, lies curled up in bed with head retracted and Kernig's sign may be present. The condition occurs so commonly in toxic children that it can hardly be regarded as suggestive of pyelitis. There is one symptom, however, which, when it occurs, must be regarded as almost pathognomonic. Rigors are an exceedingly rare condition in childhood. Their place is usually taken by convulsions. When a rigor occurs at this stage of life, pyelitis in the vast majority of cases is probably the cause. But even in pyelitis they are not common and are seldom pronounced. They more often occur as slight shivering attacks. Fever with no localizing signs associated with shivering attacks should make one at once think of pyelitis.

There are no symptoms to suggest a urinary involvement. Pain with and frequency of micturition are very seldom present and, if they are, they suggest more an accompanying cystitis. Abdominal pain is quite a common symptom. It is spasmodic and colicky in type and the child may resent abdominal examination. Pain, however, in infants or young children is often extraordinarily difficult to localize, for example I have frequently seen earache diagnosed as abdominal pain.

[1] Read at a meeting of the Victorian Branch of the British Medical Association on July 6, 1927.

Fever with nothing obvious to account for it is a thing that is met with constantly in childhood. In connexion with this the possibilities of pyelitis must always be borne in mind. The diagnosis does not obtrude itself. It has to be sought for. If this fact is remembered, the diagnosis is easily made.

The appearance of the urine is quite characteristic. It is hazy in appearance and is not clear when filtered. A drop under the high power of the microscope is seen to contain numerous motile bacilli and a varying number of pus cells. If urine obtained in the ordinary way is amber clear, then without a microscope one may exclude a pyelitis. If however such urine is hazy and contains pus cells and organisms, a definite diagnosis of pyelitis should not be made unless such findings are confirmed by a catheter specimen. Often pus and organisms are seen in urine passed into the ordinary chamber or in stale urine infected from the vagina while catheter specimens from the child as quite clear. I have seen several mistaken diagnoses made by this means. The urine is invariably acid in reaction. The course of pyelitis is very variable. Many cases often abort quickly in a few days, some take weeks. One patient under my own care ran a temperature of 40·5° to 40° C. (105° to 104° F.) from November of one year till May of the next, although the diagnosis was made and treatment was started within twenty-four hours of the onset. The cessation of fever, however, is not coincident with the disappearance of pus and organisms from the urine. Pyobacilluria may persist for days, weeks or years afterwards and recurrent attacks of fever are by no means uncommon.

So far I have spoken of pyelitis as a more or less primary condition. It is, in addition, a very frequent complication to many other conditions in childhood. The most frequent of these are gastro-intestinal infections. Quite commonly during the course of infectious diarrhœa in babies it is noted that the intestinal condition seems to be definitely improving and the stools are approximating to normal, yet a continuous fever is present. In a large percentage of these cases if the urine is examined it is found to contain pus and organisms. However, many conditions not gastro-intestinal in origin are often complicated by a pyelitis. They often produce a diarrhœa of parenteric origin which may act as the predisposing factor.

Treatment.

The treatment in the febrile stage is simple. This consists in giving alkalis in sufficient doses to produce and maintain alkalinity of the urine. This sometimes requires massive doses. Potassium citrate is the drug usually used. My own practice is to use both the potassium and the sodium salt. I usually start with 0·75 gramme (twelve and a half grains) of the citrate of potash and 0·45 gramme (seven and a half grains) of the citrate of soda every two hours till urine is alkaline, then every three hours and finally every four hours. I also order red litmus paper for the mother to test the reaction of the urine and the drug is given in sufficient quantity and at such intervals as will maintain the urine

alkaline. Such treatment is depressing and is also liable to produce a diarrhœa and a good deal of abdominal pain. This tendency is lessened if the mixture is given freely diluted with water. Pyelitis is also probably the only disease in childhood in which a child should be wakened from sleep for its medicine. The alkalinity of the urine must be maintained throughout the whole twenty-four hours. The drug in doses thus estimated must be given during the whole of the febrile period and for some time after the fever has subsided. If its administration is discontinued too soon, recurrences are liable to occur. However, opinions differ as to the best mode of treatment of these patients and the various forms of urinary antiseptics have their advocates. My own opinion is that hexamine and similar types of urinary antiseptics are quite useless during the febrile stage. I would even go further and say that if they cleared up a urinary infection then such infection was not due to the colon bacillus. In the big majority of cases response to the alkaline treatment is obtained, at any rate as far as constitutional symptoms are concerned.

However, it has to be realized that the end of the fever is not the end of the trouble, as we have the persistence of the pyobacilluria for a varying time. In quite a fair percentage of cases it clears up and gives no further trouble, but in many it does not. At times we get recurrent attacks of pyelitis, or it may act as a focus of infection for a wide range of ailments. The clearing up of a persistent pyobacilluria is often one of the most difficult of problems.

At this stage I would like to quote a few cases of my own.

The first is that of the baby whom I have already quoted. Her infection started at the age of five months and for seven months she ran a high continuous temperature. She is now twelve years of age and is still passing huge quantities of pus and in addition has developed a gross *diabetes insipidus*. Both ureters have been catheterized and the condition is bilateral. She is considerably undersized and ill-developed and of a highly unstable nervous equilibrium. More recently she has developed a generalized rheumatoid arthritis. Every known treatment has been tried with no avail.

Another case was that of a girl with persistently unsatisfactory general health in whom nothing localizing was found except a pyobacilluria. The urine gave a pure growth of colon bacilli. I treated her on the usual lines with no success. One day I found while she was in pain, a swelling on the right side of the abdomen and this determined me to have a ureteral catheterization performed. This was done by Dr. Julian Smith and he obtained perfectly clear urine on the left side. Nothing was at first obtained from the right, then during abdominal manipulation a sudden gush of pus-laden urine was obtained. Operation revealed a kidney which was practically a bladder of urine and pus and its removal resulted in a cure. It was this case, which happened a good many years ago, that taught me the advisability of this method of examination in all cases in which pyobacilluria persists.

Another case was that of a girl, aged two and a quarter years. Two months before I saw her she had an attack of diarrhœa and a few days afterwards she suddenly became ill, very toxic and had severe abdominal pain. She ran a very high temperature, at times 42·2° C. (108° F.). When I saw her she was extremely irritable and much emaciated and the diagnosis had not been made. Her urine was loaded with pus cells and colon bacilli. Alkaline treatment was pushed, but the child died suddenly a fortnight afterwards. *Post mortem* both kidneys were found to be enlarged and scattered throughout their substance were multiple small abscesses about the size of a pin head, giving a pure growth of colon bacilli. I regarded this case as a primary pyelitis and subsequently a pyelo-nephritis.

I have quoted these three cases as examples of different sequelæ. Fortunately all are not so tragic. I have followed patients for years in whom no apparent harm seemed to result from a persistent pyobacilluria; others did not come up to the normal standard of health although nothing was very definitely wrong.

The treatment of this condition is very unsatisfactory. Citrates or any alkalis are useless. I always true a course of urinary antiseptics with variable results. Some have cleared up, but I always feel they probably would have done so without. The urine at this stage is often very offensive and hexamine will certainly clear this up. The effects of autogenous vaccines in children have been most disappointing; I cannot recollect a single case in which I could prove them to be of value.

The routine I adopt, is alkalinization of the urine in the febrile stage and for some time immediately afterwards. Then a course of hexamine with acid phosphate of soda is given and in older children this is supplemented by a course of autogenous vaccine. In the very young children vaccines seemed to me to do more harm than good.

If there is no response to treatment then I refer them to the surgeon for ureteral catheterization, first, to see if the condition is bilateral and not a surgical kidney, as in the case I have quoted, secondly, if the condition is bilateral, for a course of injections of the silver salts into the pelvis of the kidney.

At the Congress in Melbourne in 1923 Dr. Gordon Craig spoke most enthusiastically of this latter method of treatment. Since then Dr. R. Bridge has published an article in THE MEDICAL JOURNAL OF AUSTRALIA in which he thinks that the beneficial effects are due to the passage of the catheter removing kinks and obstructions in the ureter and that the silver salts have little to do with it.

In two of my cases this form of treatment has produced such a pronounced degree of nervous instability that I have had to have it stopped. In others I must confess I have seen more failures than successes. Perhaps I have not persisted long enough.

I am sorry to conclude my remarks on such a pessimistic note, but hope that those who follow me, may be able to deal more optimistically with the subject with advancing years of the patient.

PYELITIS IN THE ADULT.[1]

By BALCOMBE QUICK, D.S.O., M.B. (Melb.), F.R.C.S. (Eng.).
Honorary Surgeon Attending In-Patients, The Alfred Hospital, Melbourne.

THE infection of the kidney which usually goes by the name of pyelitis, is one which has given rise to

[1] Read at a meeting of the Victorian Branch of the British Medical Association on July 6, 1927.

much discussion and towards a better understanding of which a great deal of experimental work has been carried out in recent years.

It must be admitted, however, that obscurity still surrounds many aspects of the subject and opportunities for exact pathological studies in man seldom occur. From a survey of experimentally produced infections it would seem, however, that any attempt to differentiate sharply between pyelitis and the more widespread infection of the kidney known as pyelonephritis is only misleading. While clinically we may be able to distinguish the diseases to our satisfaction, it is doubtful if any real difference exists but that of degree. From the viewpoint of a rational therapy it is obviously of importance that as clear a conception as possible should be held of the method of invasion of the kidney, its extent and the pathology of the condition.

Method of Invasion.

It has long been recognized that microorganisms, particularly bacilli, may be excreted in the urine in large numbers, as in typhoid fever, without giving rise to any demonstrable signs of inflammation in the urinary tract, that is, a pure bacilluria; it is generally held that the organisms are carried to the kidney by the blood stream in such cases. Cohnheim and others have claimed that the normal kidney is permeable within from fifteen to sixty minutes to bacilli injected intravenously. Helmholtz and his coworkers have not been able to confirm these results. They find that bacilli do not appear in the urine until a number of hours have elapsed and local effects have been produced in the kidney, with a consequent breaking down of the renal barrier. All their efforts to produce a true bacilluria in an undamaged kidney by intravenous injection have failed. Such injections result, however, in a considerable proportion of the experiments in the production of a pyelitis or pyelonephritis, particularly when the cultures used have been derived from a case of human pyelitis or, as in one series, from a pyelonephritis occurring spontaneously in a rabbit. Support is given to the theory of blood-borne infection by the analogy of those undoubtedly hæmatogenous coccal infections of the kidney which affect particularly the cortex, giving rise to abscess formation there and by extension to the surrounding fatty tissues to perinephric abscess. In a considerable proportion of such cases a clear cut history of some preexisting superficial infection, boil, carbuncle or whitlow, can be obtained and no alternatives to the blood-borne route can be entertained.

It must not be forgotten, however, that other pathways for the possible carriage of infection to the kidney exist: (i) *viâ* the lymphatic network which connects the large bowel and the kidney, (ii) *viâ* the urinary passages, urethra, bladder and ureter, as one type of ascending infection and (iii) from the bladder *viâ* the lymphatics of the ureter, an ascending infection by another route.

Though the possibility of it undoubtedly exists, it has not been found feasible to produce experimentally a renal infection by way of the lymphatics uniting large bowel and kidney, but on many occasions both types of ascending infection have been brought about by intracystic injection of colon bacilli into the unobstructed bladder.

The lesions in experimentally produced infections of the kidney in rabbits vary widely, ranging in degree from those which we conceive to be present in man in "pyelitis" to the more widespread changes typical of pyelonephritis—cortical or medullary abscesses—and it is of great interest and perhaps of some significance to find that the type of infection tends to vary with the route employed. Thus, following intracystic injection and an ascending infection the characteristic finding is an infiltration of the parietal wall of the pelvis; only occasionally is the substance of the kidney affected. On the other hand, following infection by the blood stream, only two rabbits out of twenty with pyelitis failed to show some additional involvement of kidney substance, either cortex or medulla.[1]

It would seem from a consideration of such experimental findings that the conception of pyelitis as a blood-borne infection producing an inflammation limited to the mucous membrane of the pelvis must be abandoned and with it any attempt to differentiate sharply between a pyelitis and a pyelonephritis, that is a renal infection with abscess formation in medulla or cortex. Such an alteration of viewpoint may help materially to explain the failure which frequently attends treatment directly aimed at the pelvis of the kidney. The conception of pyelitis as an inflammation of a mucous membrane alone without accompanying renal involvement is not unlike that which long confused the pathology of acute suppurative periostitis in regarding it as an entity distinct from the underlying bone infection in the metaphysis.

Ætiology.

The infecting organism in the great majority of cases is the colon bacillus and it is not very unusual to obtain a history of bowel derangement preceding the onset of the disease. This may be either an attack of diarrhœa or of constipation, presumably leading to the passage from the bowel into the blood stream of organisms in unusual numbers or of increased virulence.

Mention must be made of the common incidence of pyelitis after operation, particularly upon the abdomen. Various factors may combine to predispose to this—a general lowering of resistance, the administration of purgatives and enemata, handling of the bowel or the passage of catheters There can be little doubt that the arrest of circulating organisms by the kidney with destruction *in situ*, as opposed to filtration into the urine, is of constant occurrence and that some such upset of the balance as just indicated determines the onset of an infection. That a lowering of kidney resistance by the chronic trauma of a calculus very commonly leads to infection by the colon bacillus is well recognized. It is quite conceivable that subtle bacterial trauma may similarly lower renal resistance. Very arresting papers on this aspect have been published by Bumpus and Meisser.[2] They regard *Bacillus coli*

as a secondary invader, the primary infection and damage being due to a *Streptococcus viridans* derived from foci in teeth or tonsils, having a selective affinity for renal tissues but dying out early, supplanted and overgrown by the colon bacillus.

The disease is found occurring at all ages and in both sexes and displays, particularly in its acute form, an extraordinary variation in the severity of its symptoms dependent in all probability in large part upon the degree of parenchymatous involvement. One or both kidneys may be involved and in the latter case it frequently happens that the second becomes affected during an apparent convalescence from that first concerned. The right side is said to be rather more frequently affected and certainly it would appear so to the surgeon who is often called in owing to some suspicion of an intraperitoneal lesion on that side. In the male the infection may further involve prostate, vesicles or epididymis by surface spread apparently. I have seen perinephric abscess follow acute hæmatogenous pyelonephritis of coccal origin, but never the usual bacillary infection.

Symptoms.

As already mentioned, the symptoms of acute pyelitis or pyelonephritis vary greatly in severity and are in general well recognized. At times a history is given of vague ill-health for some days before anything definitely referable to the urinary tract is noticed. Then frequency of micturition begins with more or less malaise and prostration. It is not very rare for a definite hæmaturia to be noticed at the outset. Micturition is painful and frequent with at times a slight terminal staining of blood. The urine is acid, hazy (though this feature may not be very noticeable) and often with characteristic odour. The microscope reveals pus cells, red cells in varying numbers, motile bacilli and at times casts. Some complaint may be made of aching in the loin and tenderness in the costo-vertebral angle. Blood examination reveals a more or less well marked leucocytosis.

In the more severe cases prostration is more pronounced, both renal and abdominal pain is experienced and muscular rigidity may be found not only of the loin but of the abdomen. The tongue tends to become dry, a rigor occurs and is repeated. In such cases the condition is undoubtedly an acute pyelonephritis and the outcome may be fatal with increasing prostration, vomiting, emaciation and suppression of urine.

Diagnosis.

Diagnosis is not difficult as a rule, if the urine be examined. Failing this some intraabdominal inflammation may be suspected on account of the muscle rigidity, particularly when the right kidney is affected. Left-sided lesions present no difficulty in most instances. At times some confusion may be caused by an initial hæmaturia. A microscopical examination of the urine having been made (of a catheter specimen in women) and the infection in the urinary tract recognized, only one other condition is at all likely to simulate acute pyelitis. This is an acute colon infection of the prostate, also hæmatogenous in origin, of which several instances have come under my notice. In such there is an absence of all loin pain and tenderness and a rectal examination at once furnishes the diagnosis.

Prognosis.

The prognosis as regards life is good and I have not had experience of a fatal case. In the rare hyperacute infections or long drawn out acute illness a timely nephrectomy alone may save life. The vast majority recover without operative interference of any kind.

The febrile period of the illness in a pyelitis (or pyelonephritis) of average severity extends over two or three weeks, but convalescence is not uncommonly interrupted by one or more relapses or by an infection of the opposite kidney. The condition may then clear up completely, may persist in chronic form or may apparently clear up to be followed by recurrence in after years.

Winsbury White[3] brings forward considerable evidence to show the relationship between long-standing mild pyelitis and the pelvic type of hydronephrosis in which no gross and obvious cause of obstruction and dilatation can be made out.

Treatment.

There are certain general measures about the value of which there can be little difference of opinion. The primary source of the bacilli is the large intestine and so in most cases an initial purgative is wisely given; for this I prefer calomel in small divided doses. Thoughout the illness the bowels should be carefully regulated. As a first measure of relief large hot applications to the loin give great comfort and should be continued as long as any pain persists.

Less general assent may be given to the value of free diuresis from the ingestion of water in any and every form, but to my mind this is of the greatest benefit. Fluids should be pushed to the limits of the patient's capacity, from three and a half to four and a half or even five and a half litres (approximately three, four or five quarts) being given in the twenty-four hours, the normal output of urine being thus more than doubled. In this way not only are toxins effectively eliminated in urine and sweat but, in addition, painful tenesmus is much relieved by the dilution of the urine.

While no known drugs have any specific action in pyelitis, I believe nevertheless that the majority of patients do best under medicinal treatment and medicinal treatment only.

Treatment by alkalinization of the urine by citrates or bicarbonates, even though quite empirical, is undoubtedly of value and many investigations have been carried out in an attempt to place it upon a basis. Very little success has attended these. It has been shown that the colon bacillus will flourish in media of an alkalinity equal to the greatest observed in human beings (pH 8·6). Neither does it appear that the organisms are any less toxic when grown in such a medium. Experiments *in vitro* fail to show any appreciable lag in the rate of multiplication following alteration of the

reaction of the medium from acid to alkaline. The general symptoms of pyelitis, however, certainly seem to be improved by alkalinization of the urine and the bladder irritability to be lessened and most observers agree as to its value, at any rate in the acute stages. A routine dosage is unsatisfactory—citrates or bicarbonates or both must be administered in amount sufficient to make the reaction of the urine definitely alkaline and this amount may be quite large. Thus thirty grammes (one ounce) or more of citrate may need to be administered daily in divided doses, the effect being controlled by litmus.

It is my custom to give alkalis throughout the febrile period of the disease and for a few days after, then to allow the reaction of the urine to become acid and to begin the administration of hexamine. I give 0·6 gramme (ten grains) every six or eight hours. Some bladder irritation may result if the urine become at all concentrated, therefore the fluid intake should still be liberal.

If the urine remains neutral or alkaline, acid sodium phosphate is given in a separate mixture until the reaction is definitely acid. Large doses up to eight grammes (two drachms) every six hours may be necessary. If diarrhœa be caused by these large doses, dilute nitro-hydrochloric acid may be substituted in part. The urine of course must be acid for the liberation of the formaldehyde upon which the action of hexamine depends, and which is probably of value in diminishing the pus and organisms in the urine. I am inclined to the belief that the action of this urinary antiseptic is more in evidence in the bladder than in the kidney and that its effect is mainly upon the cystitis which often forms part of the disease. The formaldehyde may cause considerable bladder irritation, especially if hexamine be given in too large amounts or too early and not very uncommonly a return of fever appears to coincide with the acidification of the urine, compelling a return to alkalis. Apart from any effect due to the antiseptic it is possible that some "lag" does attend the alteration of reaction from alkaline to acid. The limitations of any urinary antiseptic, particularly such a one as hexamine, dependent as it is upon an acid medium for its activity, are evident. Any parenchymatous infection will be quite unaffected and it is unreasonable to look for definite results in such cases.

The urine excreted during the administration of "Hexyl-Resorcinol" in suitable dosage is quite definitely bactericidal, but its utility is subject to precisely the same limitations.

If the view be accepted that the more severe infections are accompanied by parenchymatous lesions, it is logical to seek for some remedy which, given by the intravenous route, may directly reach or affect the organisms multiplying in the cortex or medulla. From thoroughly reliable sources brilliant results have been reported following the use of mercurochrome intravenously in desperate cases of pyelonephritis. I should not hesitate to use it in a suitable case, but up to the present opportunity has not arisen.

Pelvic Lavage and Other Operative Treatment.

Special methods of treatment should not be allowed to intrude too much in such a discussion. Briefly, I hold that there is only one therapeutic indication for ureteric catheterization in acute pyelonephritis—excessive renal pain. In such cases it is possible that some swelling of the mucous membrane may obstruct the pelvo-ureteric junction or that it may become kinked by swelling of the kidney with resultant distension of the pelvis. The passage of a catheter to the pelvis may be successful in relieving this and at the same time lessening toxæmia by the catheter drainage.

Except for this one indication I believe that any catheterization and lavage is unwise. In the first place the passage of the cystoscope may cause a good deal of pain and reaction, especially in the male and secondly because I believe that patients with infections of the purely pelvic type will come through their illness as well without such intervention, whilst if the parenchyma be involved it is useless.

On rare occasions intense toxæmia and prostration with continued rigors will necessitate some major intervention, nephrotomy or nephrectomy, usually the latter. My experience of this grade of infection is limited to one case in which removal of the kidney was successful after several weeks of illness with steady deterioration.

Chronic Infections.

The chronic infection of the kidney, be it pyelitis or pyelonephritis, which only too commonly follows the acute illness calls for some remarks. Bladder irritability is an outstanding feature of this condition which gives rise upon the whole more to annoyance and discomfort than to invalidism. Such conditions, especially if the history of an originally acute attack be lacking or forgotten as it not uncommonly is, are frequently labelled cystitis as soon as a urinary examination has revealed the presence of pus and organisms.

The need for full cystoscopic investigation in such cases is obvious and no contraindication exists. It should be remembered that a primary chronic cystitis, apart from infection and reinfection from above or incomplete emptying of the bladder from any cause, is of rare occurrence. Cystoscopy and catheterization of ureters will usually show that there is a source of chronic infection in one or both kidneys, commonly with bladder pathology as well, the so-called pyelocystitis.

In a small minority of cases the urine from both kidneys is found to be sterile, although perhaps containing a few leucocytes. The explanation of such, usually occurring in women, is probably that the renal infection has died out or is in abeyance and that some local cause such as a cystocele keeps up the bladder infection. When the urine has the characteristic fishy smell, the kidney will be found to be definitely and as a rule extensively infected.

A tendency exists in these cases of chronic pyelitis to small "flares-up" from time to time. Such exacerbations with renal or ureteric pain are frequently diagnosed as appendicitis.

That a complete cystoscopic examination should precede treatment goes without saying and such an examination may involve not only ureteric catheterization with microscopic and bacteriological examination, but pyelography and functional tests. With comparatively normal renal findings further examination during an exacerbation of symptoms may be of value. The need for expert X ray examination must be stressed particularly; not uncommonly a renal or ureteric calculus is the underlying cause of the renal infection.

Treatment.

The treatment of such chronic cases is in my experience full of difficulties and disappointments. In the first place since the condition frequently causes more discomfort than anything else the subjects of it are seldom willing to submit to repeated instrumentations. They are very willing, however, to undergo treatment which consists of drugs and vaccines, probably one reason to which the latter owes its vogue. I have never been quite able to convince myself that I have seen any definite benefit from vaccine treatment. Even its advocates admit that it may need to be continued for many months, perhaps a year, before cure is attained and the patient is likely to become discouraged long before this. All are agreed that the vaccine must be autogenous on account of the great number of strains of the colon bacillus found in renal infections.

The efficacy of drugs in bringing about an abiding cure is in my opinion open to grave doubt. Hexamine will improve some patients greatly. Others are made much more comfortable by citrates. In the great majority a lurking infection persists to awaken to renewed activity at some later date.

Topical Treatment.—In a few cases in which it seems that the renal infection has died out or is quiescent (the urine from both kidneys being found sterile) whilst a trigonitis or areal inflammation still persists in the bladder, bladder washing and instillations may undoubtedly give great relief. When definite infection is present in the kidney treatment by renal lavage has many advocates. My own experience of lavage is both limited and disappointing. I have used "Collargol," nitrate of silver and mercurochrome and have never succeeded in obtaining results that might fairly be ascribed to the medicament. In those cases, however, in which attacks of renal pain have been a subject of complaint relief may be experienced. This relief is in all probability due more to the dilatation of a fibrotic ureter and improvement in pelvic drainage than to the effects of the lavage itself. The necessary and repeated instrumentations are unacceptable to the patient unless the improvement is immediate and thus treatment is apt soon to be discontinued. Moreover it is by no means a rare experience to note a definite "flare-up" following the filling of the pelvis, however gently this be done. When some lowering of health is caused by the renal infection, a greater inclination may exist on both sides to persist with the treatment. Unfortunately it is probably true that it is in just such cases that lavage is of little value. For when finally such a kidney is removed it will be obvious that infection and the changes consequent upon its long continuance involve parenchyma as well as pelvis—a chronic pyelonephritis—and that conservative treatment was foredoomed to failure. In short I would say that when a persistent primary renal infection can be demonstrated, only two courses of action appear to be satisfactory; in the mild cases the administration of urinary antiseptics, usually hexamine in the colon infections, with a repetition of ureteric catheterization and lavage if benefit seems to have resulted by relief of renal pain. Unless the condition is of many years' standing, a search for sites of focal sepsis in teeth or tonsils is well made and appropriate measures taken. In those other instances of the disease in which the toxæmia of chronic pyelonephritis is definitely impairing the general health and the other kidney can be demonstrated as free from infection and functionally reliable, nephrectomy is the wisest treatment.

References.

(1) Henry F. Helmholtz: "The Pathological Changes in Experimental, Ascending and Hæmatogenous Pyelitis," *The Journal of Urology*, October, 1922, page 301.
(2) H. C. Bumpus, Junior, and J. G. Meisser: Collected Papers of the Mayo Clinic, 1920, page 197.
(3) Winsbury White: *The British Journal of Surgery*, October, 1925, Volume XIII, page 247.

PUERPERAL PYELITIS.[1]

By ROBERT FOWLER, M.D., F.R.C.S.,
Honorary Gynæcologist, The Alfred Hospital, Melbourne; Honorary Gynæcologist, Austin Hospital, Heidelberg, Victoria.

Introduction.

MY contribution towards tonight's discussion concerns pyelitis as met with in gynæcological and obstetrical practice. It appears that this phase of the subject requires especial consideration because the incidence of pyelitis is infinitely heavier in females than in males and further because the disease is prevalent as a puerperal[2] complication.

The statistical proof of these assertions for the State of Victoria is difficult to adduce in a direct manner. As a cause of death and as a hospital diagnosis pyelitis is reported and classified in Group 131 of the International List (1920). This is a large group containing many miscellaneous items, although it is safe to say that "surgical kidney" and "pyelitis" predominate to such an extent as to comprise practically the entire group. On this assumption we obtain indirect evidence of the relative sex incidence of pyelitis from the fact that the Public Hospitals in Victoria for eleven years (1916 to 1926) dealt with 1,298 cases among males classified as "Group 131" and with 2,270 similar cases in females. Bearing in mind the relatively

[1] Read at a meeting of the Victorian Branch of the British Medical Association on July 6, 1927, and illustrated by a series of pyelograms to demonstrate the frequent occurrence of pyelo-ureteric distension in cases of frequency without manifest urinary disability and its origin at the point where the ureter crosses the psoas ridge. The pyelograms were submitted for publication, but time and space rendered this course impossible.
[2] Puerperal (*puerperus* = child-bearing) signifies an accompaniment of child-bearing, irrespective of whether such appertains to the antenatal or postnatal period.

large preponderance of "surgical kidney" in males the corrected incidence of pyelitis in females would be greater than that shown by the foregoing figures.

As Dr. S. W. Ferguson has told us tonight, in children of the napkin-age pyelitis is practically unknown amongst boys. Further evidence is obtained from the following statement:

Pyelitis at the Melbourne Hospital for twelve months 1923-1924:

Female patients	58
Male patients	13
Total	71

(Females = 81·7%)

Pyelitis at the Alfred Hospital, Prahran for twelve months 1926-1927:

Female patients	70
Male patients	18
Total	88

(Females = 79·5%)

Pyelitis (puerperal) at the Women's Hospital for eighteen months 1926-1927.

(i) *Ante partum* (indoor patient's only)—

Total admissions, all causes ..	1,356
Total admissions, pyelitis ..	64

Incidence of pyelitis = 4·7%.

(ii) *Post Partum* (indoor patients only)—

Total parturitions	4,392
Total with pyelitis	64

Incidence of pyelitis = 1·46%.

Statistics such as these, however, illustrate but a minor proportion of cases. Except for its trick of recurrence, the average case of pyelitis is adjudged a minor malady and does not find its way into either the mortality tables or the public hospital reports. Lacking therefore full statistical recognition, it remains for the general body of practitioners to register their clinical impressions in support or rebuttal of my own and other observations. Indeed, I can imagine no better subject for some future British Medical Association symposium, the triennial organization of which was suggested by our President some time ago.

Ætiology.

The disproportionate sex incidence and the puerperal liability to pyelitis are features of the disease the contemplation of which at once brings us to grips with the problem of ætiology. Indeed no theory concerning the manner and nature of pyelocystic infection can become established, unless it explains this peculiar sex and puerperal predisposition.

In view of the fact that the preponderance in females is so great we cannot, I think, escape the inference that the comparative brevity of the female urethra accords to microbic invasion a freedom of access to the urinary tract more usually denied in the male. Frank Kidd, in speaking of gonorrhœa, pronounces upon the female urethra thus: "However great are the disadvantages and disabilities of being a woman, nevertheless in one anatomical feature the woman rejoices, namely in the possession of a short and simple urethra. . . ." Nevertheless in the case of urinary infections with the *Bacillus coli* advocates of the ascending route of infection will see in Kidd's remark a sardonic solecism, since to their view the brevity and location of the female urethra expose the unfortunate possessor to a not infrequent inoculation with this putrefactive parasite.

In the pathogenesis of pyelitis, however, there are several ætiological factors other than the anatomical one just suggested. Of these I am sure one of the most important is the factor of urinary stasis. To my mind it is because of the predisposition of pregnant women to this phenomenon that puerperal pyelitis is such a common occurrence. Distension of the upper urinary tract in puerperal pyelitis is the usual radiographic or *post mortem* finding. Necropsy and pyelography may also demonstrate definite urinary distension during a pregnancy uncomplicated by urinary infection. It occurs to me therefore to urge the thesis that stasis is an antecedent and predisposing factor in the genesis of puerperal pyelitis.

The explanation of this stasis must be sought in the physical and functional modifications of the urinary tract attributable to pregnancy. Among the physical modifications we should note (i) the distortion of the bladder (especially during the latter months), (ii) the increase of abdominal pressure with consequent general compression of the ureters and (iii) the possible strained interlocking of the ureter and uterine artery due to a reorientation at their crossing points.

With the aid of a diagram we can appreciate the details of the vesical distortion produced by the filling of the lower uterine segment. In the middle line the anterior and posterior walls are closely applied to one another. This deformity results in the approximation of the vesical orifices of the ureters, rendered patulous by stretching, to the internal meatus of the urethra. This proximity might facilitate inoculation of the ureters *viâ* the urethra. On either side of the lower uterine segment the bladder extends laterally to such a degree that two deep diverticulæ are formed. Frequently they are found filled with urine after voiding. This residual urine is uncommonly difficult to remove with a catheter, as no doubt many of you have experienced in attempting to do so prior to the obstetric forceps operation.

In the case of *multiparæ* a further vesical deformity may exist in the form of cystocele. Its importance as a cause of residual urine and bacilluria has been emphasized by Rothwell Adam and others. The more advanced degrees of female genital prolapse are responsible for an extent of urinary back-working equivalent to that produced by prostatic enlargement in the male. The sections of *procidentia uteri* (Sydney University Museum) prepared by F. A. Maguire, illustrate the mechanics of urinary obstruction in such cases.

Professor Berry's frozen section (Melbourne University Anatomical Museum) shows well how cramped for room are the ureters in the presence of a large pregnant uterus. I am by no means sure that we can afford to scoff at the old theory of uterine pressure on the ureters even though we can readily pass a ureteric catheter in these cases.

Lastly the possibility of interlocking of ureter and uterine artery (after the fashion of an aberrant renal artery) is a fanciful theory worth considering. Even though no organic obstruction is thereby produced, it is a possible cause of irritative spasm of the ureter at this point.

In addition to the foregoing physical disabilities and in order to explain more completely the urinary stasis of pregnancy it is necessary to postulate a functional derangement of the normal emptying mechanism of the upper urinary tract. As will be sen later it is by no means an entirely hypothetical idea that pyelo-retention results from functional as well as organic interference with pyelo-ureteric dynamics. Indeed, it is probable that the functional form is the more frequent.

The physiology of the neuro-muscular mechanism of the urinary bladder has been long and patiently studied and we are familiar with the main features of vesical retention and distension. This may occur by reason of atony of the bladder wall or from obstruction in the urethra, whether functional or organic. We know also that a distended bladder very soon means first a bacilluria from inoculation of stagnant urine and next a pyuria from consecutive infection of the mucosa (cystitis).

Thanks to the researches of Fey and Truchat (1926) fluoroscopy of the pyelo-ureter (analogous to the familiar gastro-duodenal fluoroscopy) has revealed the normal dynamic behaviour of this portion of the urinary tract as well as certain of its aberrations. It has been shown for example that pyelo-ureteric retention and distension can occur from atony of the intrinsic musculature or from ureteric obstruction either organic (calculus, ligature, kinks *et cetera*) or functional (spasm, congestion).

Pyelo-retention originating from any of the usual causes may be an accidental association of pregnancy or on the other hand it is conceivable that the pregnancy is directly responsible for its inception.

Before reaching my objective, namely the presentation of a connected statement of a workable idea of the sequence of events in the pathogenesis of puerperal pyelitis, it is necessary briefly to divert your attention towards bacteriological considerations of ætiological significance.

The bacterium responsible for the vast majority of cases of pyelitis is the *Bacillus coli communis* apparently in pure culture. In the small minority are found other motile bacilli which are not lactose-fermenters (paratyphosus *et cetera*), whereas rarely in mixed culture occur pathogenic cocci. In conversation with me Dr. C. H. Mollison has attested the accuracy of these statements with reference to cases at the Women's Hospital, Carlton, and in Melbourne generally. It is abundantly clear from the literature that the same bacteriological conditions hold true abroad.

Bawtree (1911) has done some work which on bacteriological grounds definitely indicates that in bacillary inoculations of the urinary tract the source of the invader is the patient's own colon.

Some observations of Dr. Penfold (verbally communicated) appeal to me as strikingly relevant to the matter under discussion. If urine, fractionally voided into sterile vessels, is incubated at body temperature, a luxuriant bacterial growth occurs in the various samples at successive intervals according to the priority of voidance. If catheter specimens are similarly treated, they also after a period of some six to eight days give rise to similar growths. Specimens of urine obtained by acupuncture of the peritoneal aspect of the healthy bladder of laboratory animals remain sterile upon incubation indefinitely. The inferences we may draw from these observations are: (i) that urine is an excellent culture medium for certain bacteria (*Bacillus coli* grows luxuriantly), (ii) that it is almost impossible to avoid contamination of the urine during any transurethral manipulation.

Another fact we should note is that *Bacillus coli* is not usually pathogenic and requires enhanced virulence or reduced immunity to become so. Even when pathogenic it very rarely causes a septicæmia by heavy invasion of the blood stream, so that its excretion by the kidney must also be unusual. Such is not the case with *Bacillus typhosus*, *Bacillus paratyphosus* and *Streptococcus pyogenes*.

We may now summarize the sequence of events in the pathology of urinary infection. The initial disability is a urinary stasis throughout part or whole of the urinary tract. Such a condition may occur quite apart from gestation and even in males, but pregnancy increases the frequency of its occurrence. The pyelo-distension of pregnancy appears to be considerable, insidious and painless, attributes indicative of inertia or atony of the pyelo-ureteric musculature. Acute urinary retention is usually characterized by a localizing pain appropriate to the affected portion of the tract.

Such a condition of stasis may be relieved spontaneously without bacterial invasion occurring, but an accumulation of urine at body temperature forms an ideal culture medium for the incubation of several bacterial varieties. These may be introduced from below or by renal excretion from the blood. For reasons already stated, however, by far the most frequent urinary inoculations in womenfolk are with *Bacillus coli communis* admitted *per urethram*. In these ascending inoculations desecration of the sterility of the upper urinary tract may be the result of progressive bacillary colonization of static urine *in vivo*, although another mode of ascent is possible, namely by means of regurgitant eddies from the bladder in the presence of an ineffective sphincteric control of the ureteric meatus. Such a phenomenon has been demonstrated by various observers using coloured solutions in the bladder with a ureteric catheter in position (quoted by Luys). This second stage is the stage of bacilluria clinically characterized by an offensive decomposing urine. Frequently also acute toxæmic symptoms arise from absorption of decomposition products retained in sequestered body channels (compare lochiometra with associated sapræmia).

The next stage consists in actual tissue invasion, that is true infection. This may be general or

patchy throughout the urinary tract, so that we get such descriptive terms as pyelitis, pyelonephritis, ureteritis, cystitis. Admittedly the dividing line is a fine one and often impossible to draw, so that we speak broadly of urinary infection. The chief clinical characteristic is pyuria associated with the general signs and symptoms of infection. In the present state of our knowledge we can only speculate as to what factors determine the location of the infection. The traumatism of parturition undoubtedly determines some cases of cystitis and terminal ureteritis. Possibly miliary necroses in the renal parenchyma due to circulating streptococci derived from other foci (for example dental) may assist in the causation of the more serious pyelonephritis.

Clinical Features and Diagnosis.

Assuming the truth of the foregoing propositions it is neither profitable nor possible to consider the symptomatology of pyelitis as a detached entity until we review the clinical features of the morbid processes which prelude its inception.

By a recitation of gynæcological and obstetrical case histories I shall now endeavour to sketch in this clinical ensemble.

Case I illustrates *post partum* vesical retention with consecutive bacilluria.

Mrs. L., a *primipara*, was twenty-two years of age. Antenatal examination of urine showed it to be normal. Uneventful low forceps delivery occurred with "twilight sleep." The patient was allowed to go eighteen hours without voiding urine; she had suprapubic pain and tumour. A litre and a half (fifty ounces) were drawn off with a catheter. She was unable to do without the catheter for six days. Offensive urine with bacilluria originated on the second day. Diuresis was induced with potassium citrate. Boric bladder irrigation was instituted. Spontaneous micturition and normal urine returned on the seventh day. The only residual disability at the end of four weeks was frequency of micturition from trigonal irritation.

Case II illustrates gross painless pyelo-ureteric distension in normal pregnancy.

Mrs. P., aged thirty-two years, is now at the eighteenth week of her sixth pregnancy. She has no general obstetrical or urinary symptoms. A casual pyelogram shows gigantic right pyelo-ureteric distension (the left side was not injected).

Case III illustrates the same features as Case II.

Mrs. F. is a *primigravida* at seven and a half months. She has no symptoms. A casual pyelogram reveals right pyelo-ureteric distension.

Case IV illustrates the same features as Cases II and III.

The patient is a *multipara*, now five and a half months pregnant. A casual pyelogram reveals distension.

Case V illustrates the same features as Cases II, III and IV.

Mrs. McD., aged thirty years, a *secundigravida*, was eight months pregnant, without manifest urinary disability. Pyelography demonstrated marked bilateral pyelo-distension. Her previous pregnancy was terminated on account of intractable pyelitis.

Case VI illustrates intermittent acute pyelo-retention from probable spasticity of the lower part of the ureter due to myoma in proximity.

Mrs. M., aged forty-one years, was a *nullipara* and had been married for three years. Attacks of classical right ureteric colic (loin to groin pain) occurred during the last six months. Normal urine and no disability were discovered on skiagraphy. A myoma the size of a billiard ball was found in the right broad ligament. Passage of a ureteric catheter relieved the attack. On the pyelogram no pyleo-distension was apparent.

Case VII illustrates similar features after hysterectomy.

Mrs. M., aged forty-two years, had a pan-hysterectomy performed eighteen months ago (menorrhagia due to *fibrosis uteri*). Occasional attacks of classical left ureteric pain occurred several times during the past eight months. No abnormality was detected in the urine. The diagnosis was probable intermittent pyelo-retention from irritation or stenosis of lower end of the left ureter by scar tissue.

Case VIII illustrates pyelo-ureteric distension and bacillary inoculation of urine. No true pyelitis was present.

Mrs. F., aged twenty-one years, was a *primigravida* of five months. Attacks of pain were present in the right iliac fossa radiating towards the groin. She thought that she might have appendicitis. The urine was offensive and intense bacilluria was present, but no pyuria. No pyrexia was present. Radiography demonstrated marked right pyelo-distension. Passage of a ureteric catheter relieved the right renal pelvis of twenty cubic centimetres of urine. The pain disappeared. Slight recurrence of pain and bacilluria occurred five weeks later.

Case IX illustrates classical pyelitis. A cure resulted in six days with potassium citrate given by the mouth. Recurrences took place later.

Mrs. H., aged twenty-two years, was a *primigravida* five and a half months pregnant. Sudden onset of pains occurred in both loins, shivers and occasional vomiting were present. She had noticed cloudy offensive urine for some weeks previously and frequency of micturition.

On examination, pyrexia was present (39·4° C. or 103° F.). Tenderness was present in the right costo-vertebral angle. Pyuria was present with motile bacilli in the urine. Citrate of potash was freely given. She was confined to bed and light fluid diet was given. All symptoms disappeared in seven days except residual bacilluria for an indefinite period. Subsequent normal parturition took place.

Recurrence of frequency, bacilluria with a few pus cells, right loin pain and mild pyrexia occurred fourteen months and two years later. Skiagraphy revealed a normal tract. Cystoscopy revealed mild trigonal congestion. Rapid subsidence under citrate of potash administration occurred.

The fever produced by this class of urinary derangement is characterized by a high pyrexia of remarkably abrupt onset and a drowsy intoxication, both of which suggest the absorption in abundance of putrefactive ptomaines rather than an excessively virulent infection. I recollect one case which bore out this contention rather well in that I was able to reduce the patient's temperature to normal and to clarify her mental condition by passing ureteric catheters and draining off fifty cubic centimetres of stinking urine from the pelvis of her right kidney and a somewhat less amount from that on the left. On three or four occasions at intervals of a few days this was successfully repeated only to be succeeded by relapse within twenty-four to thirty-six hours of withdrawing the catheters. Termination of pregnancy was then undertaken with rapid and complete cure (*vide* Case V).

The differential diagnosis of appendicitis presents considerable embarrassment when it is recollected that puerperal pyelitis is commoner on the right side, that pregnancy tends to cause exacerbation of obsolescent appendicitis and that both pyelitis and appendicitis may coexist.

An amusing instance of this latter possibility was afforded by a case at the Women's Hospital several years ago.

The patient was admitted under my care with a diagnosis of pyelitis complicating pregnancy. I nevertheless suspected subacute appendicitis and asked Dr. Tate Sutherland to see her. He concurred with the original diagnosis of pyelitis and deferred operation. I thereupon made a detailed urinary investigation, observed some patches of cystitis and recovered pus in the right kidney urine. Two days later unaware of my findings Dr. Tate Sutherland again saw her, reversed his diagnosis and performed an immediate operation at which he removed a subsiding but acutely inflamed appendix.

Case X illustrates *post partum* pyelo-cystitis.

Mrs. N., aged forty years, had had her fourth child after an interval of seven years. A certain amount of genital prolapse (cystocele) was present, urinary frequency occurred and offensive urine was passed in the antenatal period. It was necessary to pass a catheter several times during the first few days of the puerperium. On the eighteenth day a sudden onset of pyrexia occurred with right loin pain, pyuria and bacilluria. She was placed in hospital on fluids and citrate of potash. On the twenty-fourth day, after amelioration of symptoms, cystoscopy revealed "cotton wool" patches in the vicinity of the vesical orifices of the ureters and also other patches of cystitis. Accumulated urine containing pus and organisms was drawn off from both kidneys. Pelvic lavage with a solution of boric acid and silver nitrate instillation produced a cure except for trigonal irritation (due to cystocele).

Case XI illustrates very chronic relapsing *post partum* pyelo-cystitis.

Mrs. M., aged sixty-four years, gave a definite history of probable pyelitis in her last confinement. At intervals of two or three years since then she had attacks of urinary frequency and pain at the end of micturition. Offensive, cloudy urine was passed. She was under my observation during the last two attacks. Patchy cystitis and pyuria were noted (no genital prolapse was present). The right ureter admitted a Number 6 catheter. The left ureter was stenosed near the vesical orifice and refused a Number 5 catheter. With both kidneys dye-excretion was delayed. Skiagraphy revealed nothing abnormal. "Caprokol" and residence in the tropics cured the condition. Bladder washes were used when the patient was in Melbourne.

Case XII illustrates the need for caution in accepting a diagnosis of idiopathic pyelitis without special investigation to rule out calculus, renal tuberculosis *et cetera*.

Mrs. X. had had six children previously and was nearing the eighth month of a twin pregnancy. Pyuria, loss of weight and anæmia of some weeks' duration were present. No response to potassium citrate and pelvic lavage occurred. Her condition was regarded as obstinate, but simple pyelitis and the pregnancy was terminated by vaginal Cæsarean section. The twins lived, but the mother died four days after delivery. *Post mortem* examination disclosed a shell of the right kidney containing many calculi. Skiagraphy, cystoscopy, pyelography and dye-excretion tests would obviate such sources of error.

Treatment.

Prophylaxis.

The best insurances against an attack of puerperal pyelitis are (i) a vigorously dynamic unobstructed urinary tract, (ii) a sterile urine protected from bacterial contamination (whether ascending or descending) and (iii) undamaged tissues backed by an effective immunity mechanism. Let us review our methods in the light of these desiderata.

Antenatal methods: The painless idiopathic pyelo-distension of pregnancy being a "silent" phenomenon is scarcely amenable to treatment. An active, hygienic existence and natural diuresis by means of liberal potations are indicated. A drug for the production of urinary peristalsis would be useful. Acute painful pyelo-retention should be relieved by the passage of a ureteric catheter during attacks and the cause then investigated.

As possible causes of enhanced virulence of *Bacillus coli* and of ascending inoculation the following may require attention, namely vaginal discharge, constipation, hæmorrhoids, fissure-in-ano, colitis and chronic appendicitis. As foci for blood-borne infection (streptococcal) we should eliminate dental, tonsillar and paranasal infections. Should a bacilluria develope, it should be treated vigorously by aqueous and drug-induced diuresis, with alkaline diuretics in an endeavour to inhibit bacillary growth by an unfavourable reaction of the urine.

The oral administration of so-called urinary antiseptics may be tried. "Urotropine" requires an acid urine and is irritating to the urinary tract. "Caprokol" is expensive; its accurate usage demands a diminution of the fluid intake during its administration. A more effective method of urinary antisepsis is by pelvic lavage, but it suffers from the inconvenience and difficulty of ureteral catheterization.

The beneficial effects of pelvic lavage accrue not only from the antiseptic action of the chemical employed, but from an astringent action on the ureteric mucosa promoting better drainage and from the release of residual urine through the catheter.

Although the prismatic or indirect method of cystoscopy usually meets all requirements during pregnancy, a peculiar difficulty is present during the last few weeks owing to the antero-posterior limitation of space in the trigonal region. To overcome this the direct method of Luys is well worth practice.

Intranatal methods: A spontaneous and easy parturition is accompanied by the minimum of traumatism to the bladder and lower portions of the ureters. Dystocia, instrumental delivery and indiscreet use of the vesical catheter all increase the hazard of *post partum* urinary infection.

Postnatal methods: The strict ritual of perineal after-care and the routine alkalinization of the urine will reduce appreciably the incidence of *post partum* pyelitis (Women's Hospital staff report). The prone posture assumed for several hours a day may also assist by relieving urinary stasis.

Curative Therapy.

The only drug of proven value is potassium citrate in large doses. Its action is dual in that it flushes the renal pelvis and makes the urine alkaline. It is usually efficacious in relieving acute symptoms, but residual bacilluria and recurrence not uncommonly follow its use.

Chronic and relapsing cases probably become such on account of lingering urinary stasis. It is quite a simple matter to measure the extent of hydronephrosis and hydroureter in these cases by draining off the retained fluid through a ureteric catheter. The normal capacity of six to ten cubic centimetres may be exceeded by fifty to one hundred cubic centimetres. As a corollary it follows that adequate drainage of the renal pelvis should be a prominent factor in the treatment of these cases. Large potations in conjunction with posture may be sufficient or recourse may be had to ureteral catheter *à demeure,* to nephrostomy or to interruption of the pregnancy. Great improvement follows drainage by ureteric catheter, but may be maintained only as long as it remains in position. Pelvic lavage with boric lotion, followed by instillations of 2% silver nitrate solution or of 10% "Argyrol" solution is a logical adjunct to the use of the ureteric catheter. There is no doubt that in the majority of cases, this method is permanently effective if repeated two or three times a week for two weeks or so, but the process is troublesome and the technique a little difficult.

Nothing is so rapidly and permanently effective in reducing hydronephrosis as emptying the uterus. Induction of labour after the thirty-sixth week (when the chances of the child are as good as at term) is often the best possible treatment. Occasionally we are forced to risk the child by induction earlier than this when large doses of potassium citrate and pelvic lavage have failed to relieve alarming symptoms.

Nephrostomy and nephrectomy have been resorted to by some surgeons, but appear to me to be the refuge of the desperate.

Specific vaccine and serum therapy have not had the success theoretical considerations promised and must be considered of doubtful value.

Acknowledgments.

For statistical information I am indebted to the Victorian Government Statist and to Dr. W. L. Carrington and Dr. George Bearham.

Access to clinical material has been kindly allowed by Dr. C. S. Wood, Honorary Obstetrician at the Alfred Hospital.

I should like to thank these several gentlemen for their help and you, my most indulgent audience, for so patiently permitting my transgression of the time limit.

THE CALCIUM AND PHOSPHORUS CONTENT OF THE BLOOD IN FRACTURES.

By G. V. RUDD, M.Sc. (Melb.), A.A.C.I.,
Bio-Chemist of the Institute of Pathological Research, Royal North Shore Hospital of Sydney.

THE conditions governing the process of ossification have been the subject of numerous investigations resulting in the proposal of several theories to account for the phenomena observed. Robison and Soames[1] [2] have shown that when rhachitic bone is immersed in a solution of calcium hexosemonophosphate or calcium glycerophosphate of sufficient concentration, calcium phosphate is deposited in the bone. Extracts of the bones, teeth or ossifying cartilage of animals are found to hydrolyse potassium hexosemonophosphate and potassium glycerophosphate solutions of pH 6·8 to 9·9, the optimum pH being 8·4 to 9·4. In general the hydrolytic activity decreases with the age of the animal. It is concluded that a phosphoric esterase is present in bone, teeth and ossifying cartilage, being secreted in the region of the osteoblasts and hypertrophic cartilage cells. Kay and Robison[3] have demonstrated that the acid soluble organic phosphorus of the blood is only partially hydrolysed by the bone enzyme and further[4] that most of this hydrolysable fraction is not hydrolysed by an extract of muscle which exhibits definite enzymic activity towards hexosediphosphoric ester. They conclude, therefore, that the portion of the organic phosphorus of the blood which is hydrolysed by the bone enzyme "satisfies the important condition of a specific substrate in the scheme of ossification." Martland and Robison[5] showed that although the greater part of the acid soluble organic phosphorus of the blood is in the corpuscles, small amounts which are hydrolysed by bone phosphatase are in the plasma. The same workers,[6] studying the bones of a human fœtus and of infants, found the enzyme only where ossification was proceeding and believe that its secretion is a phase of ossification dependent upon cellular activities.

Shipley, Kramer and Howland[7] deny the necessity to suggest the existence of an enzyme to cause the liberation of inorganic phosphorus from the red blood cells. They found that the calcification of rhachitic bone placed in solutions containing 0·03% sodium bicarbonate, sodium chloride $^{n}/_{10}$, magnesium sulphate 0·0016 normal, calcium 5·0 to 10·0 milligrammes per 100 cubic centimetres and inorganic phosphorus 1·5 to 9·0 milligrammes per 100 cubic centimetres proceeds most rapidly when the concentrations of the calcium and phosphorus are nearly those of normal serum. The addition of egg-albumin to the solutions delays calcification. When rhachitic bone is placed in normal serum, calcification occurs in nineteen to forty-eight hours whilst it does not occur in the same interval of time if rhachitic serum is used. They consider that rhachitic bones do not calcify because the concentration of the inorganic bone forming elements of the blood is insufficient and not because of any inherent inability of the bone to do so. Further, the process depends on the activity of living tissue and it is impossible to explain the selective deposition of the salts of bone.

Difference of opinion exists concerning the nature of the phosphate in bone. Bassett[8] considers that hydroxy-apatite ($Ca_3P_2O_8Ca(OH)_2$) is the only phosphate of calcium that would be deposited in a stable form under the conditions of bone formation and also that calcium carbonate could be deposited simultaneously. Howland, Marriott and Kramer,[9]

making the assumption that all the carbonate is present as calcium carbonate and the magnesium as tertiary magnesium phosphate, found that the calcium phosphate of all bones, normal or rhachitic, is tricalcium phosphate.

Wells,[10] dealing with ossification, believes that the calcium salts must be deposited from the blood which probably contains calcium in the form of tribasic calcium carbono-phosphate ($P_2O_8Ca_2H_2 2CO_2(CO_3H)_2Ca$) as suggested by Barillé. The calcium of blood serum is thought by most investigators to be partly in a diffusible and partly in a non-diffusible condition. Cruickshank[11] using compensation dialysis found the diffusible calcium to be 60% of the total calcium; and Cushny[12] found 62% to 70% of the calcium of ox serum to pass through an ultrafilter. Neuhausen and Marshall[13] by electrometric methods showed that only about 20% of the calcium is present in the ionic condition.

In active rickets the calcium of the serum may or may not be reduced below normal[14] whereas the inorganic phosphorus of the blood is invariably reduced. However it may be brought about, ossification of rhachitic bone is always associated with a rise of the inorganic phosphorus. The optimum conditions for the deposition of the salts of bone in the healing of rickets and the union of fractures may be assumed to be the same, since the results of the two processes and the source of the substances taking part are the same in both cases. Therefore studies on ossification in rickets may be taken to afford information concerning the factors determining the union of fractures and the knowledge obtained in the former condition may be applied to the latter. Approaching the problem of the union of fractures from this point of view one regards the inorganic phosphorus, rather than the calcium, as the blood constituent in this group of substances likely to elucidate partly the processes occurring. In a series of estimations of the serum calcium and the inorganic and total acid-soluble phosphorus of the blood in a number of cases of fracture it has been found that only in the inorganic phosphorus do variations of possible significance occur.

Experimental Observation.

Blood was taken from patients twenty-four to forty-eight hours after fracture had occurred and again at various times during the progress of union of the fragments. A portion of the blood was allowed to clot and the serum was used for the estimation of calcium according to the method of Clark and Collip.[15] The $^n/_{100}$ potassium permanganate solution used in the final titration was prepared according to the method of Halverson and Bergeim[16] and was found to remain constant in normality over long periods. The remainder of the blood was collected in a tube containing sufficient powdered potassium oxalate to prevent clotting and was used in the estimation of inorganic phosphorus and total acid soluble phosphorus by the Briggs[17] modification of the method of Bell and Doisy, except that in the estimation of total phosphorus it was found more convenient to digest two cubic centimetres of the trichloracetic acid filtrate of the blood with one cubic centimetre of a mixture of concentrated sulphuric acid, one volume and concentrated nitric acid two volumes. After heating to fumes of sulphuric acid and cooling somewhat one cubic centimetre of 20% sodium sulphite was added to the liquid at the bottom of the tube and the contents were again heated to fuming.[1] This modification obviated the necessity of using hydrogen peroxide of doubtful purity.

The results are given in tabulated form. The average values obtained in the analysis of eleven specimens of blood taken from normal adults are given for purposes of comparison.

It is seen that in the calcium and total acid soluble phosphorus the variations are irregular and within the limits to be expected in normal individuals. In the inorganic phosphorus the tendency of the values is to fall below normal soon after fracture has occurred and then after a period of several days to rise to a figure above normal. The only exception to this occurred in the case of the patient, K.F., whose blood taken within twelve hours of the occurrence of the fracture had an inorganic phosphorus content of 3·7 milligrammes per 100 cubic centimetres. On the fifth day after fracture 3·4 milligrammes were present, after which time the figure increased to 3·7 milligrammes seven days later. Possibly in this case blood was not taken for analysis at the stage when the inorganic phosphorus was lowest.

Patient R.W. manifested an increase of 0·3 milligrammes inorganic phosphorus after an operation for freshening the ends of the fragments. Henderson, Noble and Sandiford[18] found that the average increase in twenty cases in organic phosphorus as a result of operative treatment was 0·3 milligramme.

It appears that after a fracture has occurred there is a period approximating with the early formation of callus in which the inorganic phosphorus of the blood is below the normal value. After this interval it rises to a value frequently above normal, initiating the period in which active calcification may begin. Whilst it is not suggested that the fall and subsequent rise of the inorganic phosphorus invariably coincide with or are necessary adjuncts to the two phases of the union of a fracture, it seems that a relationship may exist between these two phenomena. Interpreted from this point of view the results of Petersen[19] who has correlated the product of the calcium and inorganic phosphorus (each in milligrammes per hundred cubic centimetres) with healing or non-healing of a fracture will be seen to indicate the relationship of the inorganic phosphorus to union rather than any special virtue of the product calcium by phosphorus. Since this product is not the solubility product of tricalcium phosphate on which some believe ossification depends this interpretation of what usefulness it possesses

[1] The author is indebted to Professor Priestley, University of Sydney, for this modification.

appears to be more rational. The results reported by Petersen are in general in agreement with those in the above table that the inorganic phosphorus is lower soon after fracture has occurred than at any subsequent time.

The cases which have been dealt with in this paper are those of fracture of bones in which the attainment of union is most frequently a matter of some difficulty, and are therefore not the most suitable for a full demonstration of the hypothesis that has been advanced.

Summary.

The calcium content of the serum and the inorganic and total acid soluble phosphorus of the blood in a series of estimations in cases of fracture have been discussed.

No significance has been attached to the small irregular variations in the calcium and total phosphorus.

It has been suggested that the observed fall and subsequent rise in the inorganic phosphorus after fracture has occurred may bear some relationship to the two phases of union, callus formation and active calcification.

References.

(1) R. Robison: "The Possible Significance of Hexosephosphoric Esters in Ossification," *The Biochemical Journal*, 1923, Volume XVII, page 286.

(2) R. Robison and K. M. Soames: "The Possible Significance of Hexosephosphoric Esters in Ossification," Part II; "The Phosphoric Esterase of Ossifying Cartilage," *The Biochemical Journal*, 1924, Volume XVIII, page 740.

(3) H. D. Kay and R. Robison: "The Possible Significance of Hexosephosphoric Esters in Ossification," Part III; "The Action of the Bone Enzyme on the Organic Phosphorus Compounds in Blood," *The Biochemical Journal*, 1924, Volume XVIII, page 755.

(4) H. D. Kay and R. Robison: "The Rôle of Phosphates in Carbohydrate Metabolism; The Action of Muscle Enzyme on the Organic Phosphorus Compounds of Blood," *The Biochemical Journal*, 1924, Volume XVIII, page 1139.

(5) M. Martland and R. Robison: "Possible Significance of Hexosephosphoric Esters in Ossification," Part VI; "Phosphoric Esters in Blood Plasma," *The Biochemical Journal*, 1926, Volume XX, page 847.

(6) M. Martland and R. Robison: "The Possible Significance of Hexosephosphoric Esters in Ossification," Part V;

TABLE I.

Patient.	Fracture.	Calcium.	Inorganic Phosphorus.	Total Phosphorus.	Days after Fracture.
Average of blood of eleven normal patients ..	—	9·9	3·3	24·7	—
H.C.	Femur	10·0	2·9	24·8	1
		10·1	3·3	25·4	21
		10·0	3·1	25·0	32
		10·4	3·1	25·4	49
R.S.	Tibia and fibula	10·0	2·9	26·0	2
		9·9	3·3	23·2	9
		10·1	3·8	24·0	16
		—	3·8	26·0	28
		9·8	2·9	—	38
N.M.	Tibia and fibula	9·9	2·6	24·0	1
		10·1	4·1	24·6	14
		10·1	4·1	25·2	27
		10·3	3·1	24·6	50
		10·0	3·2	25·0	216
E.W.	Femur	9·7	3·1	24·5	3
		—	3·7	22·3	12
		10·0	3·7	23·8	19
		10·0	3·6	24·5	31
A.W.	Femur	9·9	2·8	25·0	2
		10·1	2·9	25·7	9
		10·1	3·2	25·6	27
F.H.	Femur	9·9	2·6	23·9	1
		10·0	3·8	24·7	25
		10·2	3·6	24·7	33
		10·0	3·5	25·3	53
A.L.	Femur	10·4	2·4	22·9	1
		9·8	3·1	24·8	8
		9·8	3·5	25·0	15
		9·7	3·3	25·0	29
K.F.	Tibia and fibula	9·8	3·7	23·6	1
		10·2	3·4	24·0	5
		10·0	3·6	23·6	12
		10·1	3·4	24·8	24
J.T.	Tibia and fibula	9·9	3·1	—	2
		10·0	3·7	26·0	13
		9·7	3·7	26·2	20
		10·0	2·9	—	138
R.W.	Radius, ulna and metacarpals	10·0	2·5	25·8	1
		10·1	3·5	25·9	8
		10·2	3·1	25·0	33[1]
		10·4	3·2	26·2	43
		10·2	3·4	25·8	57
W.B.	Femur (comminuted)	10·0	3·0	24·0	1
		10·0	3·6	24·7	11
		10·3	3·9	24·5	21
		10·2	3·9	24·0	29

[1] This estimation was made before operation for freshening the ends of fragments.

"The Enzyme in Early Stages of Bone Development," *The Biochemical Journal*, 1924, Volume XVIII, page 1354.

(7) P. G. Shipley, B. Kramer and J. Howland: "Studies upon Calcification in Vitro," *The Biochemical Journal*, 1926, Volume XX, page 379.

(8) H. Bassett: "The Phosphates of Calcium," Part IV; "The Basic Phosphates," *The Journal of the Chemical Society Transactions*, 1917, Volume CXI, page 620.

(9) J. Howland, W. McK. Marriott and B. Kramer: "Studies upon the Inorganic Composition of Bones," *The Journal of Biological Chemistry*, June, 1926, Volume LXVIII, page 721.

(10) H. G. Wells: "Chemical Pathology," Third Edition, page 439.

(11) E. W. H. Cruickshank: "Studies in Experimental Tetany," II; "Colloidal and Ionic Calcium," *The British Journal of Experimental Pathology*, August, 1923, Volume IV, page 213.

(12) A. R. Cushny: "The Colloid-free Filtrate of Serum," *The Journal of Physiology*, May 18, 1920, Volume LIII, page 391.

(13) B. S. Neuhausen and E. K. Marshall, Junior: "An Electro-chemical Study of the Condition of Several Electrolytes in the Blood," *The Journal of Biological Chemistry*, December, 1922, Volume LIII, page 365.

(14) J. Howland and W. McK. Marriott: "Observations on the Calcium Content of the Blood in Infantile Tetany," *The Quarterly Journal of Medicine*, July, 1918, Volume XI, page 289.

(15) E. P. Clark and J. B. Collip: "A Study on the Tisdall Method for the Determination of Blood Serum Calcium with a Suggested Modification," *The Journal of Biological Chemistry*, March, 1925, Volume LXIII, page 461.

(16) J. O. Halverson and O. Bergeim: "The Preparation of $^{n}/_{100}$ Permanganate Solutions," *The Journal of Industrial and Engineering Chemistry*, 1918, Volume X, page 119.

(17) A. P. Briggs: "Some Applications of the Colorimetric Phosphate Method," *The Journal of Biological Chemistry*, March, 1924, Volume LIX, page 255.

(18) M. S. Henderson, T. P. Noble and K. Sandiford: "Ununited Fractures with Special Reference to the Chemistry of the Blood," *The Journal of Bone and Joint Surgery*, 1926, Volume VIII, page 607.

(19) H. A. Petersen: "A Clinical Study of Ununited Fractures with Special Reference to the Inorganic Bone-forming Elements in the Blood Serum," *The Journal of Bone and Joint Surgery*, 1924, Volume VI, page 885.

A GENERAL EXPRESSION OF OPINION, BUT NOT NECESSARILY AN EXPRESSION OF GENERAL OPINION.[1]

By R. W. HORNABROOK, M.B., Ch.B. (Adelaide), M.R.C.S., L.R.C.P. (London),

Melbourne.

WHEN Dr. Aird asked me to give you an address on anæsthesia, I was at first doubtful, but on further consideration decided to do so. I chose my title before even my subject and as a result I give you my thoughts.

I not only believe that these deaths which arise under the administration of anæsthetics are preventible, but I know they are preventible.

They are the result, gentlemen, of an error of judgement—an error of judgement not on the part of the patient, but of an error of judgement on the part of the administrator or operator or a combination of the administrator and operator.

When I see an anæsthetist writing: "I prefer to look to other causes of death than myself or my anæsthetic," I feel more than sorry, for so long as we hold such opinions, for so long will we never reach that stage of perfection in our anæsthetic work to which the greatness of our anæsthetic art really warrants that the anæsthetist should reach and for so long will these unfortunate anæsthetic deaths continue to be of only too frequent occurrence.

Granted that there is something wrong with the patient at the time of the administration of an anæsthetic, that is no excuse for a death under administration; it may be a palliating circumstance, but it is no excuse. An administrator should so know his work that, even providing there is some abnormality in the patient, he should be able to counteract it and safeguard the life of his patient.

I am thinking especially now, gentlemen, of those deaths which arise during the administration of an anæsthetic say for dental extractions or postnatal and tonsil operations and which have been of far too freequent occurrence not merely in general hospital work, but also in private practice during recent years.

The greatest stain on the escutcheon of the profession today is unquestionably the occurrence of deaths under anæsthesia, especially so in the case of these minor operations and in patients who are comparatively healthy.

There is one rule and one rule above all others that the anæsthetist, if he is to succeed in his profession and if he is to safeguard the lives of his patients, must learn and that rule is: "Never gamble." He must never get away from that rule.

Every case without exception must be handled as if the patient possibly suffered from a cardiac abnormality. It is not the hearts that give us abnormal sounds, that are the chief danger and the most likely to collapse under an anæsthetic, but the hearts from which we may hear nothing abnormal. The flabby, fat hearts which *post mortem* may be perforated with the finger without trouble. These are the dangerous hearts from an anæsthetic point of view; in other words, the toneless heart, if I may so express myself.

In every case of impending trouble under anæsthetic administration there are premonitory signs of such trouble, if the administrator is keen to observe. It may be only the slightest change in colour, it may be only an alteration in the rhythm of the respirations, it may be only a slight change in their frequency or depth, but the signs are invariably there to the one who can read them, and then and at once should the administrator cease his administration and lay his mask on one side and ask himself why. Why this change? And until he is certain in his own mind that everything is alright, he must stop his administration; never under any circumstances must he gamble.

He must first satisfy himself that he can continue with safety to his patient, he must know, not guess, what is the reason for that change and must be able to counter it.

[1] Read at the Sixth Australian Dental Congress, Melbourne, August, 1927.

An anæsthetist and also the operator can never forgive himself if through any cause the failure on his part to observe leads to the death of his patient. There is no excuse. We do not know what the loss of even that one life may have meant to the community; we certainly can never know what it means to the family when such a case arises.

In all anæsthetic work it is the little things that count; methods of handling which appear trivial to the onlooker, are really essentials.

No patient should be placed under the influence of a general anæsthetic without the administrator explaining to the patient the possible or probable sensations of that patient whilst going under—the full feeling in the head or the ringing in the head and ears or even the hammering which some patients have and others do not. This hammering is certainly more apt to occur if the head is kept low instead of being well raised during administration and is also increased if the administrator does allow the patient a free supply of air during the early stages of induction. No patient, no matter what form of administration is used, should be rushed into an anæsthetic stage without air. Air is essential in all forms of administration if the administrator is not to cut off one of his safeguards towards a successful administration without any undue strain being thrown upon the heart.

Even the weakest of hearts will stand up against the gentle push method as against the rush, knock-out method. Patients should be told to breathe evenly and naturally, to permit the far away drift feeling gradually to come over them, to let themselves go as if they did not belong to themselves and above all to remember that they are only being placed into a deep sleep and that they will feel no pain and that directly the operation is over they will wake up and feel only a bit muzzy and that at no time during induction will they feel as if they are being suffocated.

When the patient gets into the dental chair or on to the operating table, he should be told to make himself as comfortable as possible. If in the chair then he should be placed in such a position that he feels he is reclining in the chair without effort. This comfort of the patient during induction is an important point, a very important one. If the patient is uncomfortable then he is very liable to have an uneven and possibly troublesome induction which in the case of a weak heart may even lead to a fatality.

If on the operating table then the patient should lie with the head raised, not lowered and in such a position as is the most comfortable one for him.

It is well to remember that the general tendency of anæsthetics is towards cerebral congestion during induction and this is more pronounced in the head low position than when the head and shoulders are raised at a natural angle By natural angle I mean an angle in which there is no kinking of the head on the neck, but a clean fair airway.

With the head raised position on the operating table and also in the easy sitting position of the patient in the dental chair, there is less likelihood of cerebral congestion which may and often does mean excitement from cerebral irritation and thereby struggling on the part of the patient. Also less anæsthetic is required in the head raised position to bring about a stage of surgical anæsthesia and this in itself is a great gain to the patient, for there is not one anæsthetic that is a tonic, and every general anæsthetic is more or less a tissue poison and liable to bring about symptoms of poisoning and changes in the tissue cells. So that the anæsthetist who can bring about and maintain a stage of surgical anæsthesia with an absolute minimum and not a maximum amount of anæsthetic, is a distinct gain.

The mere fact that a man may administer an anæsthetic to a patient and produce in that patient a state of surgical anæsthesia does not necessarily make him an anæsthetist, not by any means. It is the duty of all anæsthetists to keep down to an absolute minimum the consumption of anæsthetic and to maintain a surgical anæsthesia by this means.

It is not a question of seeing how much anæsthetic the patient will consume without killing him, but it is an essential that the anæsthetist should see with how little consumption of anæsthetic he can maintain a surgical anæsthesia.

Before commencing induction a small, soft, folded towel is placed over the eyes of the patient; this is done to exclude all stimulus from light rays falling on the eyes. The patient should be informed as to why the towel is placed over his eyes, namely, to assist him to go to sleep the more easily. Everybody is aware of the disturbing influence of light falling upon the eyes. A patient may be sound asleep in a darkened room, but directly the light is switched on or the blinds are drawn up he will generally awaken. This covering of the eyes may appear to be a small point, but that is not so, it assists towards a calm even induction and helps to lessen the quantity of anæsthetic required to obtain surgical anæsthesia. The eyes should be covered throughout the operation. If an anæsthetist knows his work, it is really quite unnecessary for him to look at or touch the eyes during any period of the administration. An anæsthetic sleep should approach in character as nearly as possible the normal sleep of the patient. The respirations should be quiet, regular and even. The nearer the anæsthetist can approach such in his administration, so much the better for his patient. When the operation is complete, the bandage is removed from the eyes and thus a stimulus given towards rousing the patient from his anæsthetic slumber.

By keeping the consumption of anæsthetic down to the absolute minimum and at the same time maintaining a surgical anæsthesia the anæsthetist not only lessens the risk to his patient, but he is also one of the main factors in a successful convalescence. A patient even after an operation of an hour or two hours should be so handled by the anæsthetist and surgeon that within a few minutes of the removal of the eye bandages he is able to be roused should it be necessary to rouse him.

It is remarkable with what a small consumption of anæsthetic a big abdominal operation can be performed and at the same time complete abdominal relaxation obtained if the surgeon has "hands" and the anæsthetist is thus able to obtain surgical anæsthesia with a minimum of anæsthetic and a maximum of air.

But the anæsthetist is not entirely to blame; some surgeons are very uncouth and rough in their handling of a patient under an anæsthetic. They appear to think that it does not matter how rough they may be, provided the patient is anæsthetized, whereas exactly the opposite is the case. A patient requires more care in handling when under an anæsthetic than when he is not under. When he is not anæsthetized, the patient has some power of resistance, some tone in other words; whereas when he is fully anæsthetized that tone is lowered. Also a rough surgeon will require a deeper stage of anæsthesia and possibly more concentration of the anæsthetic which means more asphyxiation of the patient, and an asphyxiated patient is invariably a rigid patient, no matter from what source that asphyxiation is due, whether it be too much nitrous oxide or ether or any other anæsthetic. This does not mean that all rigidity is due to asphyxiation, but it does mean that one of the main causes of rigidity may be due to asphyxiation brought about by over-concentration of the anæsthetic.

In many conditions, in fact in a great many, especially abdominal conditions, when the anæsthetist has been over-concentrating his anæsthetic and in the case of a surgeon who does not know how to use his hands, it is not more but less anæsthetic the patient requires to obtain full relaxation. Less anæsthetic and more air, less anæsthetic and less poisoning and more careful manipulation by the surgeon.

Some surgeons can place their hand through an abdominal incision and manipulate the abdominal contents with a gentleness and precision that it is a treat to witness, others are so uncouth and rough that they would give anyone the nightmare.

As showing how unnecessary trouble may arise, let me quote to you three instances that I witnessed many years ago.

A big female patient, weighing over 113 kilograms (eighteen stone) was anæsthetized in an adjoining room; she caused no trouble during induction. She was then brought into the operating theatre, lifted up on the stretcher and owing to her weight she was rather heavily dumped on to the table. That sudden concussion was enough, her heart collapsed instantly, just as if she had been struck a heavy blow upon it. In fact that is practically what did happen, that sudden shock of coming down heavily on the table was more than her fat, flabby heart could stand in her toneless condition when under the anæsthetic; in other words, she had no power of resistance. She could have been placed on the table in the same way if she had been conscious and nothing would have happened. Of course the safe thing would have been to have anæsthetized her upon the table. That patient certainly ought not to have died.

I have seen patients collapse under an anæsthetic when they have been drawn roughly and too suddenly into the lithotomy position—again the sudden blow to the heart when in a toneless condition.

I have known a surgeon who was the indirect, if not the direct cause of death in two cases many years ago, when he adopted a bullying attitude towards a young and inexperienced anæsthetist: "Put more chloroform on and get him under, how do you expect me to work if you don't get him under." Result—concentration of chloroform, sudden collapse and death.

In each one of the above cases there was culpable negligence—a failure on the part of someone, anæsthetist or surgeon or both. The fault did not rest with the patient. Granted that there was an abnormality in the patient, it was the duty of those concerned so to know their work as to be able to counter that abnormality.

There is no excuse for bad work.

In the cases given above, as in practically every case that arises today of a fatality under an anæsthetic and especially in those short operations I have mentioned, such as tonsil and adenoid operations and dental extractions there has been an error of judgement on the part of somebody, be that somebody anæsthetist, operator or a combination of the two.

Every possible step to safeguard the life of his patient should be taken by the anæsthetist and to reduce the consumption of anæsthetic down to a minimum. For this reason no operation of any duration should be undertaken without the preliminary injection of 7·5 milligrammes (one-eighth of a grain) of morphine and 0·43 milligramme (one hundred and fiftieth of a grain) of atropine to any patient over the age of twelve years and up to any age above that.

The anæsthetist should, however, never exceed the maximum dose given above, because, should he do so, sooner or later he is bound to get into trouble in the way of possible respiratory failure especially should he use chloroform to any great extent. Morphine is stimulating, sedative, depressant and it is the duty of the anæsthetist to confine his preliminary injection to such a dose as will give the stimulating, sedative effect without the depressing one and if he does not exceed 7·5 milligrammes of morphine, he can and does do so with great benefit to himself and his patient. Of course if the patient has been having doses of morphine right up to the time of operation, it is safest for the anæsthetist to confine his preliminary injection to one of 0·43 milligramme of atropine alone.

The morphine also makes the administrations of the anæsthetic much easier in that it produces in the patient a feeling of tranquility. An otherwise nervous patient will walk into the operating theatre and get on the table in a calm quiet frame of mind which anyone would have thought impossible, if he had seen the patient the day before. The induction is calm, quiet and even, there is no

excitement but just a drift into oblivion and a by no means unpleasant drift, if the patient is told to let himself fall off quietly to sleep and if no attempt is made to cut off a free supply of air.

The atropine not only lessens the salivary secretion, but it lessens also the risk of broncho-pneumonia, in fact it reduces it practically to an unknown quantity as a post-operative event and it also controls the inhibitory action of the vagus on the heart. In other words both morphine and atropine are two of the greatest safeguards and blessings that we have, from a patient's, operator's and anæsthetist's point of view and do much to diminish not only the operative but also the anæsthetic shock in that they diminish the quantity of anæsthetic consumed by the patient.

By the preliminary injection of morphine and atropine in a small dose we so reduce our consumption of anæsthetic and yet maintain a full surgical anæsthesia that post-operative vomiting is today or ought to be practically non-existent and with this great reduction in post-operative vomiting we have a much more favourable convalescence of the patient and any fear of acidosis which is often brought about by the continuous post-operative vomiting, is cut down to vanishing point.

In any dental or mouth operation such as tonsillectomy it is advisable for the administrator when the operation is in progress to count slowly to himself from one to twelve, this represents in time about fifteen seconds.

Should his patient during this period not have made any respiration, then he should not delay a moment, but should request the dentist or surgeon to let him have free access at once to the patient and he should take no refusal, but should satisfy himself that the patient does get a clear inspiratory movement.

The anæsthetist by so doing will not only safeguard himself, but also his patient. A patient cannot remain longer than fifteen seconds with safety without taking an inspiration whilst he is under the influence of an anæsthetic.

The above is a rough general rule, but if anæsthetists saw to it that they followed it, we would have fewer coroners' inquests.

I used the words above that the anæsthetist "should satisfy himself that the patient does get a clear inspiratory movement." My reason for stressing this is that the patient may make attempts at inspiration in which there is no entrance of air into the lungs. These movements are catchy in character, especially pronounced in the neighbourhood of the diaphragm and are apt to be mistaken for a clear inlet of air to the lungs, whereas, they are merely Nature's attempt to obtain air in the case of a blocked airway. They are possibly due to the lower jaw being pushed too far down or the tongue pushed backwards by the fingers of the surgeon or dentist or to a nurse's swab blocking the free entrance of air.

In conclusion, gentlemen, I have been asked do I believe in a coroner's inquest in the case of death under anæsthesia.

I have no hesitation in saying that as at present constituted I believe that these inquiries are absolutely valueless from a scientific standpoint. I do not believe that we have got one step further forward by holding these coroner's inquiries during recent years. A coroner, if he is to be of any benefit from a scientific standpoint, must have not merely legal but also medical knowledge.

As for obtaining the whole truth at these inquiries, I doubt if we ever do that. We may obtain the truth, we may obtain even nothing but the truth, but I certainly do not believe we ever obtain the whole truth and it is in that word "whole" that the distinction rests.

When I see the words *status lymphaticus* mentioned, I really wonder what is meant and I wonder if those who use those words know.

Personally I have never been able to find out what *status lymphaticus* exactly is and when I see the expression used in connexion with these cases, I wonder if it is not really a cover for ignorance and incompetency on the part of somebody.

There is one way and one way only that we can gain scientific knowledge into the true causes of these very regrettable fatalities and that is by the appointment of a board consisting of: (i) a medical man, (ii) a pathologist, (iii) a physiologist, (iv) an anæsthetist, with power to inquire fully into every death arising during the administration or under the influence of an anæsthetic and this board must deal with all cases without exception, whether arising in public hospitals or private hospitals or in private. Such inquiry should be held in private: I believe it would get us no further forward if held in public.

But the blame does not by any means rest entirely with the profession; the public is as much to blame as we are. For a bacteriological examination it goes to a bacteriologist, for an X ray examination to a radiologist, but for an anæsthetic administration the correct or incorrect method or the slightest slip in which may mean life or death to the person concerned, well "any old thing will do, provided I do not feel anything."

There is no branch of the profession in which more keen observation and power to judge and deal with the situation is required than in the anæsthetist's job.

He must know every sign, be able to interpret every sign correctly and to act on that interpretation, if he is to get through his career without mishap to his patient. When he is at his work, he has the life of his patient in his hands and it is for him to see that he carries through to a successful conclusion that trust that has been imposed upon him.

I have spoken strongly, gentlemen, but none too strongly and no word of it would I withdraw. Let me conclude with an utterance by the late Sir Frederick Hewitt: "All the leading professional bodies have expressed approval of this attempt to reduce these terrible and regrettable fatalities which seem to constitute a standing reproach to a scientific profession."

The following two letters written to Professor Osborne explain themselves.

APPENDIX.

Dear Professor Osborne,

I am forwarding to you a copy of my congress paper. What I am attempting to do is to make it as clear as possible what I think should be done in anæsthetic work.

Of course one leaves out some of those things which one ought to have done and does some of those things that one ought not to have done.

For instance, I might have explained that unless he is unable to avoid it, no anæsthetist should have his patient reclining with his face pointing directly upwards, that is, looking towards the ceiling. This position is, however, adopted on all possible occasions by some men, with the anæsthetist pulling the chin forwards in order to maintain a fair airway, or else he has a gag in the mouth and the tongue forceps on the patient's tongue, both of which actions in ordinary cases are really a sign of failure on the part of the anæsthetist to do his work properly. When a patient is anæsthetized his face ought to be turned either to the right or the left side, whichever appears to be the more natural of the two. Some patients sleep with their faces turned left and others turned right. By doing this, not only does the lower jaw not slip backwards, but the tongue also does not fall backwards and its base does not obstruct the free entrance of air to the larynx. In any case in which I have to use a gag and tongue forceps, I always feel that I have really failed in my work—I certainly cannot remember when I last had to use either, though they have been in my anæsthetic bag for years.

Also when placing a patient under the influence of an anæsthetic, it is an advantage to "talk round a patient," not to a patient or at a patient, but round him. Talking to a patient is absolutely wrong. Invariably when I am putting a patient under I talk either to the surgeon or a nurse. This is done to take the patient's attention away from himself. The patient is told that when he is going under that for a time his hearing becomes more acute than normal, that he will hear my voice very plainly and that then it gradually begins to fade away in the distance and that if he likes he can listen to my conversation as he is going under. This increasing acuteness of the hearing in the early stages of anæsthetic administration is another of the signs of cerebral congestion during induction and should always be remembered by those present in the operating theatre, such as surgeon, nurses or anæsthetist, so that nothing is said in those early stages which the patient might recall later on. The conversation can be on any old subject so long as it is not personal to the patient, the voice should be dropped slightly as anæsthetic sleep comes on. In reality it is nothing more than taking the patient's thoughts away from himself and bluffing him into a quiet anæsthetic sleep.

Yours sincerely,

R. W. HORNABROOK.

120, Collins Street,
July 18, 1927.

Dear Profesor Osborne,

Under the circumstances, one one hundred and fiftieth of a grain of atropine is ample, the circumstances being that the patient is about to have an anæsthetic administered to him. One one hundred and fiftieth of a grain of atropine will do this—it will give you enough but not too much drying up of the salivary secretion; if you administer too big a dose of atropine you get such a dry condition of the mouth and air passages that when you start to administer an anæsthetic, especially if that anæsthetic is ether, you are very apt to bring about laryngeal spasm and obstruction of the air way. Now directly you bring about such a condition, you must bring about cerebral congestion and directly you bring about cerebral congestion, you must bring about a tendency towards convulsions from cerebral irritation, that is, provided there is such a tendency and that is why the gentleman who used an overdose of atropine, that is, an overdose in conjunction with an anæsthetic and that anæsthetic ether, got into trouble and had deaths.

What you want to do and what the one hundred and fiftieth of a grain of atropine does do, is just to give you enough of that drying to lessen, but not entirely to stop, the secretion on the air passages that arises under ether administration. Also you must give just enough atropine to control your vagus, but not to inhibit it to such an extent that you get a strong bounding pulse, which is very annoying to the patient and makes him feel as if he is having a severe attack of palpitation. The one hundred and fiftieth of a grain will also keep the skin in such a condition that no heat is lost through excessive sweating, in fact, provided the patient has been properly handled, you will find that there has been no increased shock and loss of heat by sweating, even in the severest operation.

The medical profession must learn and it is very slow at learning, that you cannot give the same dosage of a drug to a patient who is to have an anæsthetic administered to him to what you can give when the patient is not going to be placed under an anæsthetic.

Take for instance adrenalin, you may give a patient a reasonable dose of adrenalin and a quite safe one, but give that same patient, if he has a flabby heart, the very same dose of adrenalin and put him under an anæsthetic especially if that anæsthetic should be chloroform, you stand a very fair chance of killing him. In other words, the enfeebled heart under the depressing effects of the chloroform will not stand up against the extra resistance of the contraction of the arterioles brought about by the adrenalin.

I am afraid that I have not much time for the man who cannot understand that you cannot, with safety, give a patient the same dosage of a drug when he is to go under an anæsthetic to what you can give him when he is not to be placed under its influence. We medical men are supposed to be scientific and intelligent, but over and over again I bump against the same thing. Why cannot I give a patient say one sixtieth of a grain of atropine and put him under an anæsthetic when he would have taken the same dose under ordinary conditions and no harm resulted. You cannot do it with safety for this reason, the action of any drug depends on circumstances and also the presence or absence of any drug with which it may be used. It is for these very reasons and knowing the men that I have to deal with, that I have cut things down to the safety limit.

R. W. HORNABROOK,
M.B. et Ch.B., M.R.C.S., L.R.C.P.

Melbourne, July 12, 1927.

Reports of Cases.

ACUTE DILATATION OF THE STOMACH.

By ALAN E. LEE, M.D. (Melb.), F.R.C.S. (Eng.),
Relieving Surgeon to Out-Patients, Brisbane Hospital.

THE recent able contribution to this journal[a] by Dr. Brian Swift concerning acute dilatation during labour and the puerperium is interesting, not only in pointing out the possibility of such a condition occurring, but, it seems to the writer, very much more so in its revelation of the absolute rarity of this condition following labour.

For if compression of the third part of the duodenum due to drag on the superior mesenteric vessels is either a primary or secondary factor in the production of acute dilatation, it would seem that the sudden reduction in size of the uterus during labour must very frequently allow descent of the intestines and cause this compression to occur.

That acute dilatation under these circumstances is so rare practically excludes mesenteric pressure as a primary factor in the ætiology of this condition and also suggests

that the at present unknown primary condition must be one that is very rarely operating during labour.

The writer would suggest that in acute dilatation of the stomach at least two classes of cases can be distinguished, one which may be called "benign" in that correct treatment will in the great majority of cases afford rapid relief and a second "malignant" class which nearly always ends fatally.

The benign type has in a good many instances actually been seen occurring in the course of an abdominal operation and in Burton Lee's paper[2] and the discussion in the New York Surgical Society[3] which followed it, the leading features of these cases were brought out.

They are usually associated with a difficult anæsthetic and air swallowing, sometimes actually noticed, is always probable. Thus the dilating medium is explained. The actual beginning of the dilatation sometimes follows some special stimulus, such as palpation of a duodenal ulcer (Wheeler).

The passage of a stomach tube, combined with manual compression of the stomach or the actual perforation of the organ, causes it to collapse and in the majority of cases no more trouble is encountered.

In cases in which no measures are taken to relieve the primary dilatation, it is reasonable to suppose that this in some way causes duodenal obstruction, this again increasing the dilatation and thus setting up a vicious circle.

That it is only the postural tone and not the contractile properties of the stomach wall that has been lost, is evident from the fact that powerful contractions, each accompanied by a severe cramping pain, have many times been observed crossing an acutely dilated stomach. These various pieces of evidence, together with a consideration of the interesting group of cases that have followed the successful wiring of abdominal aneurysms,[4] strongly suggest that it is a nervous factor rather than a toxæmic process that is responsible for the primary loss of postural tone.

The purpose of this note is, however, to draw attention to a more malignant class of cases, in which *post mortem* evidence does not suggest that any of the usual forms of treatment would have been of any avail.

The following is the history of such a patient and the literature on acute dilatation reveals a very few similar cases.

A.B., a female, aged twenty-nine years, was admitted into hospital with the diagnosis of chronic appendicitis. She had suffered from attacks of recurring abdominal pain for many years. The pain came at intervals of about three weeks and lasted about three days. The pain was confined to the right iliac fossa and was accompanied by vomiting, lower abdominal tenderness and a mild rise of temperature. She was usually obliged to go to bed, the recumbent position giving some relief. In all she had had fifty or more attacks which did not appear to be increasing in severity.

Physical examination revealed a sallow, rather unhealthy looking woman, with narrow chest and wide pelvis. Except for deep tenderness in the right iliac fossa no pathological signs could be detected.

The preoperative diagnosis was mobile caecum and ascending colon; this was confirmed at operation and Waugh's colopexy operation was performed.

In the days immediately following the operation the patient's condition was never very satisfactory. A considerable amount of vomiting occurred and this to some extent persisted afterwards, the patient vomiting small amounts about thrice daily.

As colopexy operations are frequently followed by troublesome vomiting and there was no apparent abdominal distension, no undue anxiety was felt. On the eighth day after operation, however, the patient suddenly became restless, then rapidly cyanosed and in a few minutes died.

A *post mortem* examination was performed twenty-two hours later and the following abdominal conditions were found. There was an enormous dilatation of the stomach, the organ filling the greater part of the abdomen. The dilatation persisted with gradual diminution right through the duodenum to about forty-five centimetres (eighteen inches) beyond the duodeno-jejunal flexure. Here it abruptly ceased and then through many feet of jejunum and ileum there was such an extreme degree of constriction present that the gut was little thicker than window sash cord. The lumen was completely closed, so that an impermeable obstruction to the passage of intestinal contents appeared to be present. As the large intestine was approached the gut merged into ileum of normal diameter. The caecum and ascending colon were still firmly fixed and no mechanical obstruction was here present. There was no pulmonary embolus.

In such a case as this, where the dilatation extended beyond the duodenum to a point in the jejunum beyond which an impermeable spastic contraction of the gut was present, it appeared inevitable that this contraction formed an important factor in the ætiology of the condition.

The combination of atonic dilatation succeeded by spastic contraction again infers a nervous rather than a toxæmic origin. It must be remembered, of course, that the condition occurred in a visceroptotic who presumably possessed previously an abnormal vegetative nervous system. Although in such a case as this and the few similar ones reported, no postural or other treatment could be expected to have a favourable result, yet the important fact remains that, taking large groups of acute dilatations in which the correct treatment has been applied, the mortality is not more than 7%.[5]

The correct treatment appears to be: (i) adoption of prone posture, (ii) passage of stomach tube, (iii) washing out the stomach and then applying slight negative pressure through the tube (iv) adopting measures to prevent air swallowing till the stomach has regained its tone.

References.

[1] Brian H. Swift: "Acute Dilatation of the Stomach During Labour and the Puerperium," THE MEDICAL JOURNAL OF AUSTRALIA, August 20, 1927, page 246.

[2] Burton J. Lee: "Acute Operative Dilatation of the Stomach," *Annals of Surgery*, April, 1916, page 418.

[3] *Annals of Surgery*, Volume LXIV, page 107.

[4] G. H. Colt: "Abdominal Aneurysms Treated by Wiring," *The British Journal of Surgery*, Volume XIII, page 109.

[5] William Doolin: "Acute Dilatation of the Stomach," *The British Journal of Surgery*, Volume VI, July, 1918, page 125.

Reviews.

OBSTETRICAL DIFFICULTIES.

To write with any authority on difficult labour it is necessary that the author should have had an "unlimited supply of clinical material" and "Difficult Labour" by Samuel J. Cameron and John Hewitt shows that the writers have this qualification.[1]

The first and second sections of the book deal with delayed and complicated labour, the third with operations employed in the treatment of difficult labour, the fourth and fifth which are very short sections, with the complications following difficult labour and the prevention of difficult labour, the latter being antenatal supervision.

The book contains a great deal of useful information and should be much appreciated by the general practitioner even more than by the medical student.

[1] "Difficult Labour," by Samuel J. Cameron, M.B., B.Ch. (Glasgow), F.R.F.P.S.G., and John Hewitt, M.B.; 1926. London: Edward Arnold and Company. Crown 8vo., pp. 317 with illustrations. Price 10s. 6d. net.

The Medical Journal of Australia

SATURDAY, SEPTEMBER 17, 1927.

Our Universities.

To the ordinary man the university means little more than a place where young men and women learn things that are of small practical importance, pass a few years gaily and then are absorbed into the community neither better nor worse for the interlude between schooling and industrial life. Some regard the university with reverence because the professors and lecturers are supposed to be persons with profound knowledge of an academic kind; the stateliness of the buildings and grounds enhances this sense of respect. But few people realize that the university is the true home of learning; that its function is the propagation and accumulation of knowledge. The school child is taught lessons rather to learn how to learn than to acquire knowledge. The university student is supposed to have reached the first stage of education. He is old enough to recognize the truth of the adage that knowledge is power and to learn for the sake of that power. He has the right to expect help in the university to enable him to gather information and to apply that information for his own benefit and for the benefit of mankind. The university should provide him with facilities to contribute to the general stock of knowledge. If this be the right conception of the university, it must follow that the university is the most valuable of the national institutions. As the home of education and culture its importance will be directly proportional to the number of students who avail themselves of its teaching. It must, however, be understood that the deans, professors and lecturers have a heavy responsibility, for unless the curricula are properly planned and the teaching attains a high standard, the education and culture will not reach a satisfactory level.

The three large universities of Sydney, Melbourne and Adelaide are appealing to the public for funds to enable them to fulfil their functions more adequately than they can at present. Higher education is necessarily costly and it would be fundamentally wrong to expect the student to shoulder the financial burden of these great institutions. The governments of the Australian States find millions for unproductive trade ventures, for extravagant bureaucratic departments and for unhealthy political activities. It would be unpopular if they dipped deeply into the State purse for higher education and as a result the universities are officially starved. The University of Sydney is asking the citizens of the State of New South Wales for a quarter of a million pounds. The response is dragging somewhat, but as soon as the captains of industry have learned the true significance of the university in the life of a nation, that relatively small sum will be forthcoming. Rich men with a wide vision have given of their surplus wealth to universities in every country and will continue to endow them. It is right that they should do so. John D. Rockefeller has directed that many of his millions are to be used to increase the usefulness of medical schools of universities, but he makes it a condition precedent to any gift that the country requiring assistance should demonstrate its earnestness in this connexion by contributing to the movement as generously as it can. While the authorities of our universities are appealing to the public for money to enable them to extend and improve the teaching, our governments are failing to meet their responsibilities. In addition to a liberal annual subsidy the governments should make provision from time to time for expansion in many directions. Apart from buildings, each faculty should have full equipment for teaching and research; a considerable sum should be set aside each year for the purchase of books and periodicals and sufficient staffs should be engaged to render the museums efficient for modern requirements. The remuneration of the teachers, professors, associate professors and lecturers, should be large enough to insure that the best men would be attracted and they would be able to devote the whole of their time to their university duties. It should be a condition of appointment that no professor or whole-time lecturer may accept employment outside the university.

While commending to the serious attention of the members of the medical profession the several appeals that are being made for financial aid to the universities of Australia, we would emphasize the prior claim of the three medical schools. The medical faculties are responsible for medical education and to a large extent for medical progress. This means that they have the responsibility of two of the most important tasks in the community, that of training men and women to safeguard the health of the people and that of increasing our knowledge concerning the human body in health and disease. As soon as the public and the governments realize the justification of the claims of the medical schools, it will be necessary for those in charge of these schools to reconsider their methods and to recast the curriculum, so that the people of Australia may have the full advantage of real education.

Current Comment.

EMPYEMA.

In the series of papers on empyema, read recently before the Victorian Branch of the British Medical Association and published in these pages, the incidence, diagnosis and treatment and so forth were discussed in a way which left little to be desired. As is not unusual in such gatherings, the remarks of speakers were confined to what may be called the utilitarian side of the question. Pathogenesis received but scant mention. No one raised the question as to why a patient with pneumonia becomes the victim of empyema, when a patient with apparently the same type of infection, of apparently the same virulence, escapes. The importance of this aspect of the subject cannot be overstated, for if a complete answer to it were forthcoming, it might be possible to eliminate empyema at any rate to a large extent. It will probably be impossible to eliminate empyema, until its pathogenesis is thoroughly understood. In these circumstances it will serve a useful purpose to consider a communication by Duff S. Allen[1].

Allen has studied the relationship between hæmothorax occurring at the time of intrapleural operation and empyema following a thoracotomy in a chest not previously infected. He has also carried out some experimental work and has sought to apply his findings to the occurrence of "idiopathic acute empyema" associated with or following pneumonia. He points out that empyema following uncomplicated thoracostomy or thoracotomy is a serious and all too frequent event and he believes that the causes of its occurrence after "clean" operations have not been fully recognized. The occurrence of such an empyema has been ascribed to (i) the result of open pneumothorax and its accompanying insults to the pleura at the time of operation, (ii) the result of excessive aspiration of bacteria either during or after the operation, (iii) the result of a remaining closed pneumothorax after completion of a thoracotomy. Allen states that it is not his purpose to deny the possibility of the rôle played by any one of these factors in the causation of postoperative empyema, but to emphasize the importance of hæmothorax as an ætiological factor. It is not necessary to discuss the three factors mentioned above (Allen does so but briefly) for it is obvious that they may all take a share in the causation. Noetzel has shown that when bacteria are injected into the pleural cavity after the production of an open pneumothorax, empyema follows more rapidly and more frequently than when the same amounts of bacteria are injected into the pleural cavity without the production of an open pneumothorax. Tiegel repeated and confirmed these experiments and also noted the presence of a hæmorrhagic fluid in the pleural cavity soon after the thoracotomy. Tiegel considered that this hæmorrhagic fluid was the result of the infection of the pleural cavity. Allen holds that the blood is present in the pleural cavity before the infection is established and that the fluid aids in the establishment of the infection. In other words something else in addition to the presence of bacteria seems to be necessary in the normal pleural cavity for the production of an empyema.

Allen's first set of experimental observations were made in the course of some operations for the incision of the mitral valve. He found that empyema followed thoracotomy in eighteen out of twenty-one instances. In these operations a moderate amount of soiling of the pleural cavity occurred as a result of an irregularity on the handle of the instrument. When the soiling of the pleural cavity was only slight (the fault in the instrument having been corrected) empyema occurred in only five among forty-nine animals. When the soiling was considerable, empyema occurred in every one of a series of eighteen animals. From this he concluded that soiling of the pleural cavity with blood has an important bearing on the production of empyema after thoracotomy. Other factors had to be considered. It was argued that if the empyema resulted mainly from the presence of blood in the contaminated pleural cavity, a pleural cavity into which bacteria had been injected, and to which autogenous blood alone had been added, would frequently manifest empyema. Another series of experiments was carried out on this basis. Twenty-two animals were used and a twenty-four hour culture of a hæmolytic streptococcus obtained from a patient with acute empyema was injected into the pleural cavity of the animals (rabbits). Nine of the rabbits received in addition a quantity of autogenous blood varying in amount from 0·5 to two cubic centimetres. All nine became affected by empyema. Eleven of the remain-

[1] *Surgery, Gynecology and Obstetrics*, July, 1927.

ing animals were allowed to live long enough for the development of empyema. A quantity of normal saline solution was injected into the pleural cavity of these and only three became affected by empyema. The pleura of the remaining eight was "surprisingly normal" when examined both macroscopically and microscopically. Experiments were then carried out with the pneumococcus. When pneumococcus Type IV was used, none of sixteen rabbits subsequently manifested empyema; it was found on investigation that the bacilli had lost their virulence. Six animals were tested with Type III pneumococci. Autogenous blood was injected into the pleural cavity of three of them. These became affected by empyema. Three whose pleural cavities received no blood, died of pneumococcal septicæmia, but gave no signs of empyema. With pneumococci Type I the most interesting results were obtained. The experiments were similar in nature to those with other types. Eighteen animals were used and blood was injected into ten of these. Every one of the latter became affected by pneumococcal empyema and died. Two of the remaining eight control animals died and one was found to have empyema. At autopsy it was discovered that some blood had trickled into the pleural cavity from the site of puncture of the needle. The other six were killed and none was found to be suffering from empyema.

Allen discusses the clinical significance of his observations under two headings, that of idiopathic empyema due to the streptococcus and secondly that of idiopathic empyema due to the pneumococcus. He points out that the causes of the occurrence of empyema in the course of pneumonia are usually considered to be due to the extension of the infection to the pleural surface through blocked lymphatics or to the rupture of a subpleural abscess into the pleural space. While he does not wish "to doubt the probability of either of these causes," he wishes to draw attention to the fact that an acute empyema may be the result of a spontaneous hæmothorax which has become infected. Undoubtedly Allen has shown that if virulent organisms are introduced in a suitable medium into the pleural cavity which is an excellent incubating chamber, empyema will result. In idiopathic empyema whether due to streptococci or pneumococci, there is a preceding septicæmia and in the presence of pneumonia the tissue in the close neighbourhood of the pleura, if not the tissue in actual contact with it, is teeming with virulent organisms. Allen gives no account of the general condition of the animals after the injections were made. He describes the microscopical appearances of the serosa in one instance and these are what might be expected from the introduction of septic material into so confined a serous space. The conditions found in patients suffering from streptococcal septicæmia and in patients with pneumonia are essentially different from those obtaining in Allen's experiments. In the latter virulent organisms are introduced into a normal cavity with a suitable culture medium. In the former the organisms are present in the circulating blood and if Allen's views are to be regarded as correct, it will be necessary to show that blood finds its way into the pleural cavity. Allen holds that this occurs; he refers to the fact that bloody fluid may be aspirated from the pleural cavities of patients who are becoming affected by empyema due to the streptococcus. He also points out that the greenish tint sometimes seen in the pus from pneumococcal empyemata is due to methæmoglobin or an isomer of methæmoglobin. He ignores the possibility of serum acting as the culture medium. It is probable that he would have obtained the same results, had he used blood serum for his experiments. There is no doubt that as far as post-operative empyemata are concerned, he is right in regard to the influence of a preceding hæmothorax. In regard to empyemata of septicæmic and pneumonic origin the following two concluding sentences can be accepted if the word "probability" is altered to "possibility": "In view of this experimental data, the probability is evident that idiopathic empyema is engendered, in truth, by a preceding spontaneous hæmothorax. The clinical evidence for this probability is abundant." This does not, however, answer the question put originally as to why one patient with pneumonia becomes a victim to pneumonia, when another with apparently the same type of infection escapes. All that can be said at present is that in addition to the pneumococci present in the blood some trauma (in its widest sense) to the pleura is necessary. Involvement of the pleura in the pneumonic process will supply the trauma, continued respiratory movements will aggravate the inflammation and make effusion likely to occur. If the phagocytic action of the pleura is not equal to the occasion, effusion will increase. If a blood vessel happens to become eroded, the conditions postulated by Allen will obtain and in a severe infection diapedesis of blood corpuscles with their subsequent laking will occur. In the case of an empyema resulting from a streptococcal septicæmia it will be necessary to assume the existence of a *locus minoris resistentiæ*. The whole train of events may thus be said to depend on that abstract quality—the resistance of the patient. Unfortunately there is no way at present in which this may be gauged for clinical purposes.

PYELITIS.

THE subject of pyelitis has been discussed in the pages of this journal from many points of view. Not long ago we published three papers read before the New South Wales Branch of the British Medical Association by Stephen, Brown Craig and Lee Brown. This week there appear three read before the Victorian Branch by Ferguson, Quick and Fowler. Lesions of the ureter are demonstrated with great clarity and exactness by means of the pyelogram. Unfortunately we have been compelled to withhold the publication of pyelograms in connexion with both series of papers. In both instances the time factor has had to be taken into consideration, the extra work which has been thrown on to the machinery at The Printing House by the production of the Transactions of Congress, has made the printing of art paper supplements a matter of difficulty.

Abstracts from Current Medical Literature.

GYNÆCOLOGY.

Protein Therapy in Gynæcology.

E. GRAFF (*Wiener Medizinische Wochenschrift*, May 7, 1927) discusses the value of injections of casein in gynæcological affections. The preparation used was "Caseosan." He advises against its use for puerperal sepsis and reserves its application to menorrhagia and metrorrhagia associated with inflammatory lesions of the appendages. While not a cure-all, this treatment often accelerates recovery to a considerable degree. It should not be given to patients with chronic afebrile conditions accompanied by dense adhesion formation. The best results were obtained in patients manifesting a considerable reaction. Pain is quickly relieved and palpable tumours often rapidly subside. The dosage recommended is 0·5 cubic centimetre followed by 0·5, 0·5, 1·00, 1·00 and 2·00 cubic centimetres. The interval between doses depends on the reaction induced and on an average is two days. On at least one of these days the temperature must be normal and if there be much reaction the dose should not be increased. A course should not extend beyond fourteen days, followed by an interval of at least one week. General supporting measures including regulation of the bowels should be adopted at the same time.

Tubal Sterilization.

J. HOFBAUER (*Surgery, Gynecology and Obstetrics*, June, 1927) describes a method of tubal sterilization which he has used since 1917 with uniformly good results. Pregnancy has occurred after many of the operations in common use for sterilization. Dougle ligation of the tubes with division between them has frequently failed. Either the lumen has been restored after absorption of the ligatures or a tubo-abdominal fistula has formed. The method of sterilization by burying the fimbriated ends of the tubes in the broad ligaments has not proved infallible. Moreover, this operation may prove to be exceedingly difficult on account of the vascularity of the broad ligament. An operation frequently practised is that of excising the tubes at the cornua of the uterus by a wedge shaped incision. This is certain in its results, but if pregnancy seems desirable at some future time a second operation to reimplant the tubes into the uterine cornua would probably not be successful. The operation devised by the author may be performed in two ways. According to the first method the tube is divided and the proximal end is ligated with silk, the suture being left with long ends. One end of this suture is inserted through the round ligament and the other passes through the ovarian ligament. When the ends of the suture are pulled together, the round and ovarian ligaments are approximated over the cut ends of the tube, so that they eventually envelop it in a blind *cul-de-sac*. As a further precaution one or two sutures are inserted on either side of the first suture. The principle involved in the second method is practically the same as that employed in the fixation of the inverted stump of the appendix to the caecum. The tube is severed and the proximal cut end of the tube is ligated with silk. With a fine needle a suture is inserted through the round ligament, the suture then passes through the tube, the round ligament is pulled up into a loop and the suture again passes through it. When the ligature is drawn together, the stump of the tube is buried in an artificial niche which has been produced in the posterior surface of the round ligament. As a precaution the area of peritoneum in contact with the end of the tube is scarified and an additional suture is inserted. The author claims that this operation is especially useful when merely a temporary sterilization is required and that at a second operation the liberated ends of the tubes may be united.

Radium in the Treatment of Advanced Carcinoma of the Cervix.

GILBERT I. STRACHAN (*Journal of Obstetrics and Gynæcology of the British Empire*, Summer Number, 1927) reviews the results obtained in Cardiff since 1922 in the treatment of advanced malignant disease of the cervix with radium. Ninety-seven patients with inoperable growths were treated. The author concludes that radium is a most potent palliative and in a small proportion of cases curative agent in carcinoma of the cervix. The relief of symptoms, especially hæmorrhage and discharge, is most pronounced in the majority of cases. The effect of radium on a particular case of carcinoma cannot be foretold. It may be brilliant or nil, so that prognosis always has to be guarded. There is no clinical or histological evidence that in the presence of unfavourable reaction radium has any effect in stimulating the growth of the tumour.

Pathological Forms of Corpus Luteum.

WILFRED SHAW (*Journal of Obstetrics and Gynæcology of the British Empire*, Summer Number, 1927) reviews the subject of the pathology of the *corpus luteum*. He concludes that *corpus luteum* hæmatomata are determined essentially by primary ovarian hyperæmia. Intraperitoneal hæmorrhage may be produced either by the addition of a second factor of the nature of trauma or may rise independently of this further factor if the hyperæmia is extreme. In the normal mechanism of atresia of the follicles atrophy of the granulosa cells is the rule. Sometimes they may undergo luteinization and give rise to cysts to which the term granulosa lutein cysts has been applied. Pathological forms of the follicle have been described. Such follicles are characterized by containing tarry fluid and by having lutein tissue in their walls. Three types have been described, namely, tarry theca lutein cysts, tarry granulosa lutein cysts and tarry *corpus luteum* cysts. Such tarry cysts may manifest a heterotopic endothelial lining which has been shown to arise from endothelial cells by a process of metaplasia. In old forms of such cysts the epithelium persists, the lutein cells disappear and are replaced by hyaline tissue. The tarry cysts may rupture and lutein cells become implanted upon adjacent organs in the pelvic cavity. The ætiology of the tarry cysts is unknown. They are not inflammatory in type. They may be related to ductless gland disturbances.

Cirsoid Aneurysm of the Uterus.

W. P. GRAVES AND G. VAN S. SMITH (*American Journal of Obstetrics and Gynecology*, July, 1927) describe a case of cirsoid aneurysm of the uterus. They have been prompted to do so by a report of a similar case by Dubrenil and Loubat. The patient was sixty-two years of age and was admitted to hospital on account of uterine bleeding. A normal menopause had occurred when the patient was forty-eight years of age. A diagnosis of carcinoma of the fundus was made and the patient was prepared for curettage of a diagnostic nature. Introduction of the curette into the uterus was followed by severe arterial hæmorrhage. The uterus was plugged and the abdomen was opened. Supravaginal hysterectomy was performed. The authors first described the condition as a hæmangioma, but altered the designation after reading the report by Dubrenil and Loubat. They point out that a hæmangioma is a neoplastic growth of blood vessels. It is congenital or early development. Their patient and that of Dubrenil and Loubat were sixty-two years of age and both had had a normal menopause. They think that the condition was the result of arteriosclerosis. The uterus after removal was found to contain in its wall a tortuous and irregular blood sinus 1·5 centimetres in diameter. In addition the myometrium was riddled with dilated, irregular vessels and sinuses.

Perforation of the Uterus.

E. HERZ (*Wiener Medizinische Wochenschrift*, June 11, 1927) describes various errors of technique leading to perforation of the pregnant uterus. In many cases there has not been an exact estimation of the position and size of the uterus. Frequently the cervix is insufficiently dilated before manipulations are performed. Too much force is used, especially with instruments which are sharp or pointed. A curette should be fairly large and have blunt rounded edges. Ovum forceps are particularly dangerous. Often the uterus is retroflexed and sufficient traction is not made on the cervix to straighten out such deflections. Before commencing the operation the curette should always be passed as far as the fundus and any manipulations carefully controlled by one hand grasping the fundus through the abdominal wall. An unsuitable position of the patient in

a sagging bed will also add greatly to the risk of perforation. Too little care is taken to differentiate conditions found according to the period of pregnancy. The size of the uterine cavity after the fourth month demands the greatest care possible, especially when the operation is performed at one sitting. The risk is also great with the puerperal uterus.

OBSTETRICS.

Effect of Radiation on the Fœtus.

W. WEIBEL (*Wiener Medizinische Wochenschrift*, September 11, 1926) describes a case in which extensive radiotherapy was carried out at the tenth week of pregnancy. The pregnancy had not been diagnosed at the time and the author first saw the patient at the end of the fifth month. The uterus was emptied because of the probable damage to the fœtus. Macroscopical examination disclosed no fœtal abnormalities. In another instance the patient was examined by X rays at the third month and went to term. The child is now three years old and is an hydrocephalic idiot. At least one-third of these fœtuses are damaged by X rays. In a series of thirty-six cases there were twice as many females as males. An ovary is never normal after radiation. Animal experiments prove that the greatest damage is done to the second generation.

The Involution of the Uterus Post Partum.

JOHN H. TEACHER (*Journal of Obstetrics and Gynæcology of the British Empire*, Spring Number, 1927) has reviewed the literature dealing with the involution of the uterus *post partum* and has investigated four uteri. He states that knowledge of the naked eye and of the microscopical appearances of the uterus at various periods *post partum* besides being necessary for the proper understanding of the process of involution in relation to obstetrics, may be of critical importance in medico-legal problems such as those of criminal abortion. Involution is a complex process, owing to the complexity of the organ concerned. In normal circumstances and even in the presence of grave constitutional and slight local disease, involution probably progresses at a fairly regular rate. The greatest disturbance of the healing process is due to the retention of some of the products of conception, the result of which is to cause delay. Exact chronological data cannot be given, but data sufficiently accurate to be helpful can be given and comparison with cases reported in the literature may be very useful in treatment. The most reliable information is provided by examination of the mucous membrane in the regions which were covered by the *decidua vera*, and in the placental site. Both these areas should be examined. The changes in the first week are chiefly concerned with demarkation of tissues which are to perish and be shed in the lochia. Retrogression of the surviving decidua and reactive proliferation are also occurring. In the second week the processes of cleansing and restoration advance rapidly and formation of a provisional mucous membrane is normally completed by about the fourteenth day. There is a striking analogy between the process of involution and that of a wound healing by granulation in the presence of sloughs and microorganisms. The fourteenth day is roughly the turning point at which building up definitely exceeds retrogression. At three weeks the process of restoration is still far from complete. At five to six weeks restoration should be practically complete, but evidence of recent pregnancy may be recognizable for a considerably longer period, even in circumstances most favourable to involution. Such evidence is found in the presence of pigmented phagocytes and remains of the utero-placental vessels.

Accidental Hæmorrhage.

L. KRAUL (*Wiener Medizinische Wochenschrift*, April 16, 1927) states that during the period 1921 to 1926 twenty-seven patients suffered from accidental hæmorrhage among 15,000 delivered at the First Clinic, Vienna. Thirteen hæmorrhages were severe and two were fatal, while fourteen were milder in character. The maternal mortality was 7·4%. The first fatal hæmorrhage was accompanied by considerable hæmorrhagic infiltration of the uterine wall and large extravasations into the parametrium. The other patient died from *post partum* hæmorrhage which was treated by supravaginal hysterectomy. Albuminuria was noted in 33% of the cases. As regards treatment sixteen patients had a spontaneous delivery. The membranes were ruptured in five when the *os* was at least half dilated and pains were occurring at regular intervals. Version and Cæsarean section were each performed twice. The fœtal mortality was 44%. In the majority of cases the fœtus was dead before admission of the patient to hospital. Reviewing the results obtained Kraul advocates a waiting policy in the presence of mild hæmorrhage, but considers that patients with the severe types should be delivered as rapidly as possible, due consideration being paid to the stage of labour. Hence Cæsarean section will find a place for patients with severe hæmorrhage, who are not in labour.

Unusual Indications for Cæsarean Section.

E. HERZ (*Wiener Medizinische Wochenschrift*, April 23, 1927) describes two Cæsarean sections for which the indications were unusual. The first patient was a *primipara*, aged thirty-two, with a history of scarlet fever and enteric fever in girlhood. The latter was associated with pronounced abdominal symptoms. Menstruation did not occur until the age of twenty-three. The patient was admitted with a history of being in labour for two days. The uterus was firmly contracted round the fœtus and Bandl's ring was prominent. Pelvic measurements revealed a general contracted and rickety flat pelvis with a *conjugata vera* of seven centimetres. The cervix could not be felt *per vaginam*. Following operation and the delivery of a living child the cervix was found to be above the symphysis and to be held in that position by adhesions between the uterus and the abdominal wall due apparently to inflammatory reaction following the enteric fever when the patient was sixteen years of age. The second patient was a *primipara*, aged twenty, who gave a history of a fall when six months pregnant, followed by severe abdominal pain and fever for four weeks. She had been three days in labour before admission and also had a temperature of 38·6° C.. Pelvic measurements were normal, but no *os* could be felt on vaginal examination. After operation the cervix was found to be drawn high up by dense adhesions between the uterus and the abdominal wall due to trauma following the accident during pregnancy. Both operations were of the low cervical type.

The Fluctuations in Blood Sugar During Eclampsia.

PAUL TITUS, PAUL DODDS and E. W. WILLETTS (*American Journal of Obstetrics and Gynecology*, July, 1927) have carried out some work on the treatment of patients with toxæmia of pregnancy by high carbonate feedings and intravenous injections of hypertonic glucose solution. They state that evidence is now offered by them that in eclampsia the disturbance in carbohydrate metabolism, heretofore assumed, actually exists. This is apparently the result of the carbohydrate deficiency in pregnancy which has been postulated. Hyperglycæmia is not characteristic of eclampsia. Serial blood sugar readings during an attack of eclampsia reveal wide fluctuations in exceedingly short intervals of time. Similar series in normal pregnant women near term manifest no such variation. The convulsive seizures occur at levels which the authors designate "relative hypoglycæmia," and are caused by the sudden drops in blood sugar now being reported. In this respect eclamptic convulsions are comparable to those of "Insulin" hypoglycæmia. Following a convulsion there is usually a temporary rise in blood sugar, the usual physiological response of the liver to muscular activity. There exists a tendency towards remissions to lower levels so that the general trend of the sugar content of the blood in eclampsia is downward, obviously the effect of exhaustion of reserve glycogen stores in the liver. With the view that the convulsions of eclampsia are to be designated as a hypoglycæmic reaction or manifestation, the use of "Insulin" either with or without glucose in the treatment of this disease is unnecessary and contraindicated. Intravenous injections of hypertonic glucose saline solution has now a definite basis for its proved therapeutic value.

British Medical Association News.

SCIENTIFIC.

A MEETING OF THE VICTORIAN BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held at the Medical Society Hall, East Melbourne, on July 6, 1927, DR. J. NEWMAN MORRIS, the President, in the chair.

Pyelitis.

DR. STEWART FERGUSON read a paper entitled: "Pyelitis in Children" (see page 388).

MR. BALCOMBE QUICK read a paper entitled: "Pyelitis in Adults" (see page 389).

MR. ROBERT FOWLER read a paper entitled: "Puerperal Pyelitis" (see page 393).

DR. W. H. G. CUSCADEN desired to thank the readers of the opening papers. In his gynæcological out-patient department he had seen a large number of patients suffering from chronic pyelitis. For a considerable period it had been his practice to wash out the renal pelvis with a solution of silver nitrate. He had on one occasion accidentally used a blocked catheter and had found that the result had been quite as good as in those cases in which lavage had been done. Since then it had been his practice to dilate the ureters, using as large a catheter as possible. He thought that the beneficial results were due, not to the lavage, but to the dilatation of the ureters accompanying it. By using a direct vision cystoscope instead of the ordinary indirect type, he had found it possible to pass a larger catheter into the ureters. In the use of the former instrument the wearing of a spectacle frame with a convex lens in front of one eye was of considerable help. Where trigonitis was present, painting the inflamed area with silver nitrate solution was of value.

MR. C. GORDON SHAW said that he had little to add to what had already been said by the previous speakers. He agreed with Mr. Quick that in most if not in all cases of so-called pyelitis, the kidney substance was involved. In the acute cases the main lesions were in the kidney, while in the chronic cases the pelvis was chiefly affected, although kidney changes might also be present. He believed that a pure bacilluria was very rare, most conditions so diagnosed being either pyelitis or prostatitis.

In not a few patients in whom a diagnosis of chronic pyelitis had been made, some organic disease of the kidney other than the infection was present. He instanced a girl, sixteen years of age, who had been treated for pyelitis for five months and in whom X ray examination had shown a calculus in the kidney. He had also seen several patients the persistence of whose symptoms had been due to hydronephrosis which had been demonstrated in the pyelograms. He considered that in all cases of chronic pyelitis a complete examination of the genito-urinary tract should be undertaken. In acute pyelitis instrumental treatment was not indicated and in most cases was harmful and might cause suppression of urine. In chronic pyelitis, on the other hand, local treatment was most important. White considered that in a majority of cases hydronephrosis was due to a stricture of the ureter, either organic or spasmodic, following infection of the renal pelvis. Mr. Shaw had frequently found that in chronic pyelitis the pyelograms showed slight dilatation of the renal pelvis and in some cases he had been surprised to find that the urine had been sterile, the infection having apparently died out. This suggested very strongly that dilatation was a sound form of treatment and that as large a catheter as possible should be passed with the object of dilating the whole ureter. He thought that the condition was analogous to the soft non-fibrosed stricture sometimes seen in infections of the urethra where dilatation was also effectively used.

In lavage of the renal pelvis he had at first used colloidal silver which had been strongly recommended by Kidd, but the results on the whole had been bad and the urine had still contained organisms. He had then used a 1 in 2,000 solution of "Flavine" and had found that in every case the urine had become sterile and all symptoms had disappeared after one, two or three treatments. In one case of chronic pyelitis of over twenty years' duration the urine had become sterile after two treatments and was still sterile eighteen months later. Although as a general rule he advised lavage only in chronic cases, he had had encouraging results from its use in subacute cases which had shown no signs of subsiding in three or four weeks' time. In these only one side at a time should be irrigated. He had been very much interested in Mr. Fowler's explanation of renal colic in myomata of the uterus. He had had a patient with bilateral renal colic in whom a calculus had been removed from one kidney, but colic had persisted on the opposite side and had been relieved by the removal of a myoma from the broad ligament. He had appreciated very much the opening papers.

DR. A. V. M. ANDERSON thought that the opening papers had been excellent. He was in complete agreement with Dr. Ferguson as to the great benefit of making the urine alkaline in acute pyelitis. In both children and adults a cure usually resulted, but if the infection still persisted a search should be made for some underlying cause. If a case of pyelitis or cystitis proved resistant to treatment by alkalis and hexamine, the patient should be handed over to the surgeon. He quoted a patient with cystitis who many years previously had been cured by the digital removal of a bladder calculus by the late Dr. O'Hara. In the centre of the calculus was a small piece of drain tube. The patient had had a myoma removed some time previously and Dr. Anderson thought it probable that a small piece of the drain tube which it was the custom to insert in those days, had broken off and ulcerated through into the bladder. He also mentioned a patient with long continued pyelitis of pregnancy in whom repeated rigors had occurred. It had been decided to induce labour and quinine, twelve centigrammes (two grains), three times a day had been administered. In a week the pyelitis had completely cleared up and he wondered whether this was spontaneous or due to the quinine.

DR. H. FLECKER referred to the frequency of pyelitis as a late distressing complication of uterine carcinoma. He thought that this was probably due to the extension of the malignant process to the lower ends of the ureters. When catheterization of the ureters was not possible, it had been suggested that they be transplanted into another part of the bladder or the rectum.

DR. CLIVE DISHER desired to support Mr. Shaw with respect to the good results obtained by irrigation of the renal pelvis with "Flavine" in cases of chronic pyelitis. Two patients both of whom had resisted treatment by alkalis and hexamine, had been referred by him to Mr. Shaw and in each instance a cure had resulted from the use of the above-mentioned form of treatment. A catheter specimen of urine taken from one patient five months later had been sterile. Pyelitis had occurred in five patients under his care whose bladders had been carefully catheterized in connexion with labour. Since then he had administered citrates as a routine procedure after catheterization when this had been done and had not had any further cases of pyelitis.

DR. GERALD WEIGALL said that in cases of acute pyelitis it was generally agreed that the urine should be made alkaline, but he had sometimes found this difficult to achieve owing to the fact that the patient had vomited citrate of potassium and sodium; he had not found bicarbonate of soda very effective. He asked whether in such cases there was any other method whereby the urine might be made alkaline. He also desired to support Mr. Shaw's statement that irrigation of the renal pelves with "Flavine" was successful in chronic cases sometimes even after only one treatment. In several patients referred by him to Mr. Shaw this treatment had been very effective.

DR. A. M. WILSON discussed the treatment of acute pyelitis of pregnancy. He had frequently found that citrates had caused vomiting and in these circumstances he had given sodium bicarbonate, but had not found it to be satisfactory. He had not seen any good result from

catheterization of the ureters and postural treatment was not of much value. He thought that the administration of large amounts of water, about nine litres (two gallons) a day was the most important point in treatment. He had seen a number of patients who had been so ill that the pregnancy had had to be terminated and instanced one who had had rigors for one month, in whom induction of labour had been followed by a dramatic cure. He thought that if the condition were allowed to go on too long unrelieved, irreparable damage to the kidney might result.

At the Women's Hospital it had been found that pyelitis was the cause of 15% of patients having a rise in temperature in the puerperium. By the routine administration of citrates in all cases and the elimination of the catheter as far as possible this percentage had been reduced to seven. He had seen three cases of pyelitis caused by delay in emptying the bladder after labour and in all three rheumatoid arthritis had subsequently developed. The difficulty in diagnosis between acute pyelitis and appendicitis in pregnancy was very great and in view of the fact that the mortality rate of the latter was 20%, he thought that if there was a reasonable suspicion that appendicitis was present during a pregnancy, the appendix should be removed.

DR. GEOFFREY PENINGTON advised the giving of citrates along with syrup of Virginian prune and aniseed water. He had found that since he had adopted this procedure, he had been able to administer four grammes (sixty grains) of citrate every two hours without causing vomiting.

DR. A. P. DERHAM said that in both acute pyelitis and enuresis in children he had found it much easier to make the urine alkaline, if the child were placed on a high protein diet. This might be of help in those cases in which large doses of citrates caused vomiting.

DR. ROBERT SOUTHBY agreed with Mr. Shaw that the term pyelitis should be abandoned. Pyobacilluria was a more correct description of the condition, as the urine was frequently loaded with pus cells and bacilli and yet *post mortem* examination revealed no gross change in the renal pelvis. Occasionally children were seen who had run a long course and in whom a definite pyelonephritis was found with points of pus in the kidney substance under the renal capsule.

He concurred with Dr. Ferguson that true rigors were rare in infants and he could recall only three in whom they had occurred. In two of these *post mortem* examination had revealed abscesses in the kidney substance, but no changes in the renal pelvis. In the third case decapsulation had been performed on both kidneys and these had manifested a few points of pus beneath the capsule. The patient had made a good recovery.

DR. J. W. GRIEVE had listened with great interest to the papers presented. He wished to support very strongly the administration of citrates in doses of 1·3 to 2·0 grammes (twenty to thirty grains) every two hours till the urine was alkaline and thought that the vast majority of children with acute pyelitis would respond to this treatment. The citrates should be continued for weeks or even months. In considering the method of invasion it had to be remembered that the vast majority of affected children were females. In many cases *post mortem* examination had revealed a definite pyelonephritis and in one case in which operation had been performed in the belief that a perinephric abscess was present, pyelonephritis had been found and the patient had made a good recovery under medical treatment. He thought that many patients looked upon as having pyelitis, had in reality pyelonephritis and a large number recovered. He had been very interested in Mr. Fowler's remarks on urinary stasis. He had occasionally seen children who had been regarded as having pyelitis, and *post mortem* examination had shown hydronephrosis and huge dilatation of the ureters with consequent stasis and infection.

MR. J. T. TAIT had been very much interested in the opening papers. He discussed the treatment of pyelitis, especially the chronic form in children and adults. He could not agree with Dr. Ferguson's pessimism with regard to the outlook of chronic pyelitis in children. He had treated fifty patients with satisfactory results. He instanced two patients, ten and twelve years of age respectively, one of whom had been treated for five and the other for seven years, with drugs and vaccines. In each case specimens of urine taken from both ureters had been shown by Dr. R. Webster to contain many pus cells and bacteria. Lavage of the renal pelves with a solution of silver nitrate had given good results in both instances. After the fourth treatment the urine had become sterile and free from pus cells and had remained so after eighteen months. The mental and physical condition of all the patients had definitely improved. He agreed with Mr. Fowler that in the treatment of chronic pyelitis it had to be remembered that urinary stasis was the important factor causing persistence of symptoms. Gross anatomical obstruction might be present, but in many cases there was a lesser degree of blockage due to swelling, narrowing or kinking of the ureter. It was not necessary to have any evident dilatation of the renal pelvis to cause renal pain; it was the earlier stages of dilatation that caused the most pain which might be very slight when definite hydronephrosis had developed. It was useless and even dangerous to commence renal lavage before determining whether gross anatomical changes were present or not. In acute pyelitis lavage should not be done.

DR. J. NEWMAN MORRIS said that the diagnosis and treatment of pyelitis had been laid down most clearly. Perhaps the pessimism expressed by Dr. Ferguson, in view of what had been said by some of the speakers, had not been fully justified. He congratulated the readers of the opening papers and called on them to reply.

DR. FERGUSON said that he would look forward to a trial of the treatment of chronic pyelitis as outlined by Mr. Shaw. In children, in marked contrast to adults, pyelitis usually caused no characteristic symptoms and the diagnosis would be made only if the urine were carefully examined.

MR. QUICK said that he was glad to see a recognition of the view that so-called pyelitis was in reality a pyelonephritis. The remarks of the various speakers, particularly those of Dr. Shaw and Dr. Cuscaden, showed the difficulty in interpreting clinical and experimental results. The opinions expressed by them raised the question whether the success of renal lavage was due to the drug employed or to the dilatation of the ureter.

DR. FOWLER welcomed the many helpful suggestions made by those who had taken part in the discussion. He did not agree with Mr. Shaw that uncomplicated bacilluria did not occur. An occasional pus cell obtained by the use of a very high velocity centrifuge was of no importance. He thought that in gynæcological work bacilluria without pyuria was very frequent.

A MEETING OF THE NEW SOUTH WALES BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held at the Royal Alexandra Hospital for Children, Camperdown, on September 8, 1927. The meeting took the form of a series of demonstrations by the members of the honorary staff.

Cerebral Hæmorrhage After Pertussis.

DR. R. B. WADE showed a male patient, aged four years, who had been admitted to hospital on August 20, 1927, with a history of having suffered from pertussis for one month. He had fallen out of bed on the day of admission on to his head and had been found unconscious on the floor. At the time of his admission it had been impossible to rouse him. No bleeding had occurred from the cranial orifices, the pupils had been widely dilated and had reacted to light. The knee jerks had been present and exaggerated and the abdominal reflexes had also been exaggerated. His colour had been good and the pulse soft, rapid and regular. He had not regained consciousness on the following day and it had been necessary to feed him by means of a tube. On August 22 an X ray examination had been made, but no fracture had been discovered. It had been possible to rouse him, but he had resented interference. He had vomited several times. On August 23 he had been considerably

worse, his pulse had been weak and irregular and he had suffered from attacks of alternate cyanosis and pallor. The respirations had at times been stertorous. Several attacks of twitching of the muscles of the left hand, arm, face and leg had occurred. Lumbar puncture had been carried out and thirty cubic centimetres of clear fluid under increased pressure had been withdrawn. No improvement had resulted. Eleven hours later a generalized convulsion of the left side had occurred and slight improvement had occurred when ten cubic centimetres of clear fluid under slightly increased pressure had been withdrawn. Five hours later the pulse had been irregular and the patient had been cyanosed. Cheyne Stokes breathing had been noticed and the patient had had more convulsions, localized mainly to the left side. Craniotomy had been performed, a needle had been introduced subdurally and some cerebro-spinal fluid withdrawn. The puncture had not been sutured. The wound had then been closed. Cerebro-spinal fluid had drained into the dressing for a period of thirty-six hours. The child had gradually improved and had been allowed to sit up on September 3, at this time the wound had healed. He had not spoken, but was seen to watch the movements of objects.

Congenital Right Talipes Equino-Varus.

Dr. Wade also showed a male patient, aged five years, who had been admitted on March 2, 1927, with a congenital right *talipes equino-varus*. Some interference had evidently taken place with the lower epiphysis which had ceased to grow. Relative overgrowth of the right fibula had caused a condition of *equino-varus*. The right leg was ten centimetres (four inches) shorter than the left. On March 3, 1927, tenotomy of the *tibialis anterior* and of the *tendo Achillis* had been performed as well as forcible eversion of the foot. In addition 2·5 centimetres (one inch) of the distal end of the fibula including the epiphysis had been excised subperiosteally. The fragments had been apposed and the wound closed. Two weeks later the foot had been put into plaster in a position of dorsiflexion and eversion. The child had then been discharged to attend as an out-patient.

Renal Glycosuria.

DR. E. H. M. STEPHEN showed a male child, aged eight years, who had been admitted to hospital on June 21, 1927. There were four other children in the family and all were healthy. The patient had suffered from varicella at the age of four, but had had no other illnesses. The child had been quite healthy until six weeks prior to admission when he had begun to wet the bed. Previously he had had no enuresis. Associated hunger had been present, but no increase in thirst. He had not lost weight. At the time of admission the patient had been passing more urine than usual and the mother had said that it was pale. On admission the patient had been healthy looking. The tonsils had been enlarged and had shown evidence of chronic inflammation. No other abnormality had been discovered on physical examination. The specific gravity of the urine had been 1020 and it had contained a considerable amount of sugar. On the day after admission the patient had been put on a reduced diet and his urine had been sugar free in two days. It had been found that by restricting his carbohydrates it was possible to render his urine sugar free and he had manifested no symptoms. He had been discharged on July 13 with instructions as to diet and directions for testing the urine. Since that time he had reported at intervals of a week.

Dr. Stephen pointed out that many tests had been carried out. The *sella turcica* appeared normal on an X ray film. The blood sugar on the day of admission had been 0·194% and the serum had not reacted to the Wassermann test. A glucose tolerance test had been carried out on July 1, 1927. Blood had been collected at 9·30 a.m. The urine had been sugar free at the beginning of the test, but had reduced Fehling's solution at 10.30 a.m. and at 12 noon. The following results had been obtained:

Blood sugar on a fasting stomach	0·047%
Half an hour after a glucose meal	0·141%
One hour and a half after a glucose meal	0·173%
Two hours after a glucose meal	0·132%
Two hours and a half after a glucose meal	0·115%

On July 6, 1927, the total volume of urine collected for twenty-four hours had been 920 cubic centimetres and the concentration of glucose 1%. The total amount of glucose excreted had been 9·2 grammes.

Hodgkin's Disease.

Dr. Stephen also showed a boy who was suffering from Hodgkin's disease. The child had previously been an inmate of the hospital and had been readmitted on June 21, 1927. He had given a history of cough for two weeks and of pain in the right side of the chest. On examination the boy had looked well, but a mass of glands had been present in the right axilla and some glands had also been palpable above the clavicle. The chest had been resonant, the breath sounds vesicular and there had been no accompaniments. The heart had appeared normal. On June 28 a von Pirquet test had been made, but no reaction had been obtained. The patient's temperature chart had been irregular. Bronchial breathing and slight dulness had been noticed over the right hilar region on July 5, 1927. Dr. Stephen pointed out that the patient had continued to run an irregular temperature chart, ranging from 37·2° C. (99° F.) in the morning to 38·9° C. (102° F.) in the afternoon. The glandular masses did not appear to be increasing in size, the patient's appetite was good and he felt well. Some enlargement of the mediastinal glands had been found on X ray examination, especially on the right side of the superior mediastinum. A blood count made on June 29, 1927, had yielded the following result:

Erythrocytes, per cubic millimetre	3,200,000
Hæmoglobin value	40%
Colour index	0·6
Corpuscle index	64
Leucocytes, per cubic millimetre ..	6,900
Polymorphonuclear cells	62%
Lymphocytes	19%
Monocytes	16%
Eosinophile cells	3%

Rickets and Scurvy.

Dr. Stephen also showed a child, aged one year and four months, who had been admitted to hospital on May 18, 1927, with its previous history unknown. It had been fed on "Lactogen" from the age of four months. The child had been seen to lie on its back and to toss its head ceaselessly from side to side. The skin had been dry, the head was small and the fontanelle closed. The teeth had been covered with some sordes and around the gum margins rings of purple colour, due to submucous hæmorrhage, had been found. The lower epiphyses of the bones of the forearms and the lower ends of the tibiæ were thickened and there was a definite "rosary" at the costo-chondral junctions. The skin of the abdomen was shrunken and the skin lax, the muscle tone was poor. X ray examination had been carried out and definite rhachitic changes had been found. The serum had not reacted to the Wassermann test. An X ray examination made two months after admission revealed considerable improvement. Treat- consisted of the use of ultra-violet light, "Tricalcine," adrenalin and cod liver oil.

Syphilitic Chorioiditis.

DR. N. GREIG showed a female patient, aged eleven years, who had been admitted to hospital on August 30, 1927, complaining of failing sight of three months' duration. The child was a well developed girl with a left divergent squint. The pupils were equal and reacted to light and accommodation. The knee jerks were present. Examination of the fundi revealed small white patches of various shapes and sizes with heaped up pigment around them. These changes were most pronounced in the macular region. A diagnosis of syphilitic chorioiditis had been made.

Mongolism.

DR. E. H. M. STEPHEN showed a girl, aged five years, who had been admitted to hospital on February 14, 1927. The child was backward, she had sat up at two years of age and at the time of admission had been walking for ten months. Dr. Stephen pointed out that the child

manifested the usual stigmata of mongolism. At the time of admission the child had been suffering from a severe form of erysipelas and later had had a series of boils in various situations. After the subsidence of this infection thyreoid medication had been instituted.

Peripheral Neuritis.

DR. A. W. CAMPBELL showed a girl, aged six years, who was suffering from peripheral neuritis of obscure origin. She had been admitted on August 8, 1927. She had previously suffered from pertussis. For three months she had manifested increasing paresis of both legs. At the time of demonstration she was almost completely paralysed and was quite unable to walk or stand. At no time had she complained of pain in the legs. During the previous month she had gradually lost power in the hands and arms. Considerable muscular wasting of both extremities had occurred. She had had no other symptoms. There was nothing which suggested lead poisoning or mild diphtheritic infection. The child spoke quite well and was comfortable in bed. She was intelligent. The fauces were clear, no lead line was to be seen on the gums. The pupils were equal and reacted. The palatal reflex was sluggish, but there was no regurgitation of fluids. The muscles of the trunk were unaffected, those of the extremities were very wasted and the thenar eminences were flat. The knee jerks were absent. Sensation was normal, there was no muscle tenderness and the patient had control of the sphincters. The serum had not reacted to the Wassermann test. A blood count had revealed the following information:

Erythrocytes, per cubic millimetre	3,380,000
Hæmoglobin value	80%
Colour index	1·2
Leucocytes, per cubic millimetre ..	12,000
Polymorphonuclear cells	70%
Lymphocytes	24%
Monocytes	3%
Eosinophile cells	3%

No punctate basophilia or polychrome staining had been found.

Progressive Lenticular Degeneration.

Dr. Campbell also showed a boy, aged twelve years, who was suffering from progressive lenticular degeneration. The patient was the third in the family, he had a sister living, aged sixteen years and one sister had died at the age of six months of "summer complaint." The child's birth and infancy had been normal and he had been a bright healthy boy until the age of four years when a slight stutter had been noticed. This had disappeared. At the age of five years the patient had injured his finger in a machine and this had seemed to precipitate the speech trouble again. From this onward his disabilities had gradually progressed. His mother described a stoppage in his speech, stiffness of his muscles, especially those of the right arm and leg and a peculiar gait. He was mentally backward.

Dr. Campbell pointed out that the child was a well developed boy, lying comfortably in bed. His expression was alert. He quickly grasped what was said to him, but his articulation was difficult. He made an obvious effort to answer questions at once and then replied in an explosive manner in as few words as possible. He was unable to read or write, but recognized familiar objects. He was emotional. His head was slightly smaller than normal. There was definite spasticity of the right arm and both legs, more particularly the right. He walked in a stiff fashion with a wide base. Coarse tremor was present in the right arm and leg and this was more pronounced when he attempted a voluntary movement. Muscular development and muscle power were good. Sensation was apparently normal. He had control of the sphincters. The pupils were equal and reacted to light and accommodation. The abdominal reflexes were absent, the cremasteric reflex was present. The knee jerks were present. The plantar reflex was flexor in type. No ankle clonus was present. A diagnosis of progressive lenticular degeneration had been made.

Cirrhosis of the Liver, Probably Syphilitic.

DR. M. J. PLOMLEY showed a boy, aged five years, who was suffering from cirrhosis of the liver and ascites, probably of syphilitic origin. There were four other children in the family and all were healthy. The child had been ailing for six months. He had suffered from rheumatic fever and this had been followed by abscesses of the neck, elbow and side. Then he had contracted pneumonia and the heart had become affected. Œdema of the extremities had occurred, ascites had been present and signs of congestion had become evident at the bases of the lungs. The abdomen had been tapped on several occasions. The patient's serum had not reacted to the Wassermann test.

Hydrocephalus and Achondroplasia.

Dr. Plomley also showed a child, aged thirteen months, who was suffering from hydrocephalus and achondroplasia and also from unresolved pneumonia. There were two other children in the family and both were healthy. The child had been born after a breech presentation. The condition of the head had not been noticed by the parents until the child was five months old. The child could sit up for a few minutes only. Dr. Plomley drew attention to the typical achondroplasic condition. Consolidation of the right apex and base of the left lung were present.

Pneumococcal Peritonitis with Involvement of the Appendix.

DR. WILFRID VICKERS showed two patients who had suffered from pneumococcal peritonitis with involvement of the appendix.

The first was a boy, aged four years, who had been admitted on August 1, 1927, with a history of having become ill on the previous day. He had complained of pain in the abdomen and had been put to bed. He had awakened again with pain and with his legs drawn up. He had vomited twice. The only previous illness from which the child had suffered was influenza. Considerable tenderness had been present on the right side of the abdomen with rigidity. The throat had been slightly infected. The heart and lungs had been normal. The temperature had been 38·3° C. (101° F.), the pulse rate had been 136 and the respirations had numbered 40 in the minute. A diagnosis of acute appendicitis had been made and Dr. Vickers had opened the abdomen by the gridiron incision. A ruptured appendix had been found with numerous adhesions and with free foul-smelling pus in the abdominal cavity. The appendix had been removed in the usual manner. Two drainage tubes had been inserted. One had been inserted through a stab wound and another had drained the incision. For the following four days the child had been very ill. The pathologist had reported the presence of pneumococci in the pus and injections of antipneumococcal serum had been given every day for four days. About the fifth day after the operation the patient had begun to improve. On the eighth day after operation the tubes had been removed and the patient had made an uninterrupted recovery, being discharged from hospital on August 26, three and a half weeks after admission.

The second patient was a boy, aged nine years, who had been admitted on July 15, 1927, with a history of sudden pain occurring in the right side of the abdomen the day previously. The patient had vomited several times. Examination had revealed generalized tenderness all over the abdomen, especially on the right side. The lungs and heart had been clear. The temperature had been 39·4° C. (103° F.), the pulse rate had been 120 and the respiratory rate 40 in the minute. The patient had had an operation for hernia a year previously. A diagnosis of acute appendicitis had been made and Dr. Vickers had opened the abdomen by the usual gridiron incision. A very friable appendix had been discovered, acutely inflamed with free thin yellowish pus in the abdominal cavity. The appendix had been removed and the wound closed with two drainage tubes, one into the abdomen and the other into the incision. The patient had done well after operation. The pathologist had reported the presence of pneumococci in the pus and also in the appendix. Injections of antipneumococcal serum had been given every day for five days. On the twelfth day after operation the temperature had suddenly risen and a rash had appeared. At first it had been supposed that the rash was due to scarlet fever, but later it had been found that it was only an urticarial

rash with a generalized reaction. Thereafter the patient had made an uninterrupted recovery and had been discharged on August 8, three weeks after admission.

Syphilitic Arthrosis of the Knees.

DR. F. C. ROGERS showed a male patient, aged nine years, who had been admitted to hospital on August 15, 1927, complaining of a painless swelling of one week's duration. There were four other children in the family and they were all healthy. The mother had had two miscarriages between the birth of the first and second children, but none previous to the birth of the first child. The child looked healthy and was well nourished. Examination revealed that the teeth were not of the typical Hutchinson type. The enamel was deficient at the margins of the upper central incisors. The margins were slightly grooved. No abnormality was discoverable in the abdomen. The circulatory, respiratory, renal and nervous systems were clear. The right knee joint was definitely enlarged. There was no diminution of movement or pain on movement of the joint. The right knee joint seemed to have a large effusion, more particularly in the upper part of the joint. Some thickening of the periarticular structures was present. Treatment was being carried out by means of "Neokharsivan" given intravenously and mercury and iodide of potash given orally. It had subsequently been ascertained that five months previously the patient had lacerated the right knee and that it had taken a long time to heal. A von Pirquet test had been carried out, but no reaction had been obtained. X ray examination had failed to reveal any bony lesion and the serum had reacted to the Wassermann test. So far no reduction in the size of the joint had occurred.

Pulmonary Tuberculosis.

DR. M. J. PLOMLEY showed a female patient, aged six years, who had been admitted to hospital on August 8, 1927, after ailing for fourteen days. The patient had suffered from feverishness and general malaise. A diagnosis of pulmonary tuberculosis had been made. A "+++" response had been obtained to the von Pirquet test and X ray examination had revealed consolidation of the lower lobe on the right side. The patient's father was suffering from pulmonary tuberculosis.

Congenital Dislocation of the Patellæ and Knock Knee.

DR. R. B. WADE showed a boy, aged four and a half years, who had been admitted to hospital with congenital dislocation of the patellæ and knock knee. Two other children in the family had inguinal hernia and the patient had also undergone an operation for double inguinal hernia. The patient had always walked awkwardly, falling over both backwards and forwards. He had not tired quickly, but the gait had become more awkward of late. No actual deformity had been noticed until one month previously when the knees "looked the wrong shape." It had been found that the patient could walk well without assistance. Bilateral *genu valgum* had been present and this had been exaggerated on flexion of the knee joints, when the patellæ were dislocated to the lateral aspect of the femoral condyle. On extension the patellæ had been almost normal in position. On X ray examination no shadow of either patella had been discernible. Dr. Wade had performed supracondylar osteotomy on the right femur and had placed the limb in plaster and the child in a Hamilton splint. X ray examination had shown that the fragments were in excellent position.

Mediastinal Tumour.

Dr. Wade also showed a female patient, aged seven years, who had been admitted to hospital on account of a lump which the parents had noticed in the child's throat. The family history and the child's previous history were good. For three weeks the child had had a severe cough in the early morning. No hæmoptysis had occurred. On August 20, 1927, the lump had been noticed low down beneath the sternal head of the right sterno-mastoid muscle. The lump had rapidly increased in size. Enlarged veins had also been noticed on the patient's chest. Slight local tenderness had been present on pressure over the mass in the neck. No aphonia nor alteration in the quality of the voice had occurred. There was no dyspnœa and the child had not complained of dysphagia, but of feeling tired. Examination on admission had revealed that the child was slightly cyanotic about the face, but comfortable. The voice had been clear. No laryngeal stridor or recession of the chest had been noted. She had been able to swallow semi-solids with ease. The hard, brawny swelling had extended from the left sterno-mastoid muscle across the anterior triangle at the level of the cricoid cartilage, deep to the right sterno-mastoid and about half way across the posterior triangle. The skin had moved freely over the tumour. No heat had been present, but slight redness and tenderness. Dilated veins had extended over the right side of the chest and the superficial veins of the right arm had been somewhat prominent. No œdema had been present in the face or in the right arm. The breath sounds had been equal on both sides and there had been no accompaniments. No dulness nor tubular breathing had been present. The cardiac apex beat had been palpable 8·75 centimetres (three and a half inches) from the middle line. The heart sounds had been clear. No hepatic nor splenic enlargement had been present. No generalized glandular enlargement had been noted, but the glands had been palpable. The pupils had reacted to light and accommodation. The knee jerks had been exaggerated. The patient had had a divergent squint. Five days after admission it had been noted that the tumour had definitely increased in size and that the child became cyanosed on assuming the recumbent position. She had been troubled by night cough. X ray examination had been carried out and a "huge mass" had been found in the mediastinum which was regarded as probably being lymphosarcoma. On September 2, 1927, two days after therapeutic application of X rays, it had been noted that the tumour had decreased in size, the tumour was harder and the child could breathe with more comfort.

Blood Dyscrasia.

DR. E. H. M. STEPHEN showed on behalf of Dr. Margaret Harper a child who was suffering from an obscure hæmatological condition. This case will be reported in full at a later date.

Oxycephaly.

DR. W. EVANS showed a female patient, aged three and a half years, who had been admitted on August 22, 1927, suffering from diphtheria. The child was suffering from oxycephaly. Dr. Evans pointed out that the head was very large and oxycephalic in character. It did not, however, tower antero-posteriorly. The occpital area was flattened. Slight proptosis of both eyes was present with widened palpebral fissures. The eyes were somewhat slanting. The hands and feet were deformed, all the digits were webbed together. There were no supernumerary digits. No deformity was depicted in the long bones of the extremities. The bones were not shortened. No deformity was present in the spinal column. The child seemed content and was fairly intelligent. The cranial nerves were intact. Speech was indistinct. The knee jerks were normal. The abdominal reflexes were present. The movements of the upper and lower extremities were free in range and were of usual strength. The fundi were normal, no optic neuritis or optic atrophy was seen.

Pathological Specimens.

DR. FRANK TIDSWELL showed a series of pathological specimens. He had chosen these to illustrate as far as possible the conditions which were exemplified in the several patients. The lesions demonstrated included specimens of mediastinal tumour, lymphoid hyperplasia in a Peyer's patch, lymphoid hyperplasia in a lymphatic gland near the pancreas, lung and hilum glands from a case of lymphatic leucæmia and specimens of the thymus gland. He also demonstrated the various grades of appendicitis, culminating in ruptured appendix and peritonitis. He demonstrated a section of the brain showing the internal capsule and the lenticular nucleus. He also showed several speciments demonstrating congenital lesions of the heart.

Medical Societies.

THE MELBOURNE HOSPITAL CLINICAL SOCIETY.

A MEETING OF THE MELBOURNE HOSPITAL CLINICAL SOCIETY was held at the Melbourne Hospital on July 22, 1927, MR. VICTOR HURLEY in the chair.

Spasmodic Movement of the Head.

DR. W. W. S. JOHNSTON showed a woman, aged thirty-one years, who had attended the out-patient department first on July 4, 1927, complaining of twitching of the head to the left. The twitching was not so pronounced when the patient was in bed and was absent when she was asleep; it was controllable to some extent by pressure of the hand and had first been noticed a week previously. Otherwise she was well, but had recently been working very hard sewing which had involved turning the head to the left. In the past history there was nothing suggestive of a rheumatic infection; there were three children and the patient was separated from her husband.

On examination the patient was an ill-looking woman with intermittent jerking of the head to the left, involving the contraction of the right sterno-mastoid and spinal muscles. The teeth had been badly decayed and infected and had been extracted. The heart and lungs were normal, but the temperature had been 37·8° C. (100° F.) and had remained at that level. Dr. Johnston had been inclined to regard the condition as chorea, especially in view of the temperature. He had prescribed salicylates and bromides and local treatment and had sent the patient home to bed. After a week of treatment the condition had seemed improved and the movements less frequent. Then the teeth had been extracted and the condition was definitely worse again. He favoured the diagnosis of a functional condition in view of the repetition of the same movement which was of a purposeful character. This might be associated with her occupation of a seamstress which necessitated a constant turning of the head to the left or to a circumstances elicited by Dr. Maudsley in his examination of the case. The raised temperature still remained unexplained.

DR. H. F. MAUDSLEY said that he had thought at first that the condition was functional. He had been interested to get the history that she had been separated from her husband; she had gone away and one of her friends thought she feared her husband and kept turning her head away, fearing he might recognize her. Was this a possible cause? Dr. Maudsley thought she would recover with persuasion and suggestion.

DR. A. S. M. TYMMS agreed with the diagnosis of a functional condition, as on examination no muscle or group of muscles was responsible for the twitchings.

Disseminated Sclerosis.

Dr. Johnston's second patient was a male, *ætatis* thirty, who had first attended the hospital on January 5, 1927, complaining of weakness in the legs which had become progressively worse during the previous two years. Close questioning had resulted in the following history being obtained. In 1912 he had suddenly become completely blind for one month and the sight had gradually returned. In 1913 the left eye had been affected in the same way, but the sight had returned after two weeks. He had served in the infantry throughout the war. About two and a half years previously he had noticed an inability to keep a moving object, such as a tennis ball, in sight. Subsequently he had noticed a slight stagger in his walk, twelve months previously a sensation of numbness in the left arm, left leg and eventually in the whole left side of the body. There had been no headache, but the head felt as though he were walking in a mist or were badly drugged; also he had suddenly started laughing for no reason and was unable to control this.

The patient had a spastic gait and a double extensor plantar response, the superficial abdominal reflexes were absent and the tendon reflexes were exaggerated. There was no definite sensory loss, slight Rombergism, slight nystagmus and slight intention tremor were present. Speech was unaltered and the discs showed signs of atrophy with lowered vision, especially in the left eye.

Dr. Johnston said that, although the diagnostic triad was incomplete, he thought there was little doubt as to the diagnosis. The interesting points included the long interval between the initial ocular symptoms in 1912 (if indeed these were part of the disease) and the onset of the weakness in 1924 and the fact that he had served with the infantry through the war. Secondly, there was the interference with emotional control and, lastly, the fact that he was a worker in copper. This was interesting because some authorities considered that metallic poisons were a possible ætiological factor.

Dr. Maudsley said that, as far as the emotional symptoms were concerned, the work of Wilson and others was interesting. They considered that there was a definite psychological entity typical of disseminated sclerosis. These people were not hysterical and these authors explain it on anatomical grounds. The almost invariable finding was degeneration of the paleo-thalamus.

DR. L. E. HURLEY said that he had under treatment a patient with disseminated sclerosis in whom the transitory bindness had preceded other symptoms by a period of about eleven years. He thought that in these early cases, even though the blindness might rapidly recover and no other symptoms occur for years, physical signs such as absent superficial abdominal reflexes, extensor plantar responses *et cetera* were usually present. Disseminated sclerosis, judging by published statistics, was commoner in the British Isles than in Victoria and most of the patients that he had seen recently suffering from the disease had been born overseas. He suggested that treatment with injections of "Arsenobenzol" might benefit.

DR. S. O. COWEN said that there was little to discuss in the diagnosis; if the transient blindness were the only symptom present, it would be hard to diagnose at that stage.

MR. A. E. COATES asked Dr. Maudsley if the emotional symptoms in Wilson's disease were of similar origin to those in this case.

Dr. Maudsley replied in the affirmative.

Prostatic Calculi.

DR. J. T. TAIT reported the histories of three patients who had suffered from prostatic calculi. The first was that of a man, *ætatis* sixty-four, with a history of syphilitic infection. He had complained of difficulty in micturition for years, complete retention fourteen years previously and again three weeks before admission. The urine had contained albumin and pus cells. On rectal examination the prostate had been enlarged, irregular, hard in parts and moderately tender. X ray had shown that the prostatic area was occupied by a series of small rounded shadows up to the size of a split pea and closely massed together. Suprapubic cystotomy had been done and three weeks later the general condition having improved, removal of the prostatic contents had been attempted by the suprapubic route. The patient had collapsed suddenly during the anæsthetic. At *post mortem* examination a dilated heart with damaged muscle had been found and the prostate had been found converted into a series of small sacs with tough fibrous walls in which lay numbers of small calculi.

The second history was that of a man of fifty-six years who suffered from asthma and bronchitis. He had had difficulty in micturition for twelve months, but had not suffered from complete retention. Nocturnal frequency had been present for a year; he had had to pass urine two or three times a night. There had been pain at the root of the penis during micturition. X ray examination had revealed a bilobed shadow in the prostatic area of the shape and size of a normal prostate. The medial groove had been distinctly seen. A hard, rounded mass had been felt in this region on rectal examination. A sound had passed beyond the *compressor urethræ* and had then come into contact with stone. At operation a bilobed stone had been removed with a channel through the median part. A thin shell of prostatic tissue had been cut away.

Death had occurred three days later and at *post mortem* examination congestion of the base of the right lung had been found with old pleural adhesions, poor cardiac muscle and endocarditis.

The third history was that of a man of fifty-seven years who seven years previously had had a stone removed from the bladder and at that time three calculi had been found embedded in the prostate. Two years previously he had began to have pain at the end of micturition with increased frequency and the urine had become very thick. The patient had been short of breath on exertion and had had a cough with much sputum due to extensive pulmonary fibrosis and bronchitis. On rectal examination there had been a stony, hard, mass in the prostatic region. It had been possible to pass a metal catheter into the bladder. X ray examination had revealed a bilobed shadow occupying the prostatic area and above it a large rounded shadow in the vesical area, the appearance of a typical mushroom calculus. The bladder had been opened suprapubically and the vesical part of the calculus broken off and removed. The bladder had become clean during drainage, but the patient had died four weeks later before anything further could be done. The cause of death had been septic bronchopneumonia.

Commenting on these cases, Dr. Tait said that true prostatic calculi were not very common and these three cases seemed to illustrate progressive stages in the formation and growth of prostatic stones. They arose as concretions retained within the centre of the gland as in Case I, probably a gland which was the seat of a chronic inflammation. By fusion they formed calcareous masses which replaced each lobe and united across the mid-line to produce the bilobed calculus as in Case II. Further growth resulted in projection of the stone into the bladder, where it was rapidly added to by deposit of phosphates from infected urine and the mushroom calculus was formed as in Case III. These patients had all been bad "operative risks" apart from their urinary condition. At the same time the prostatic condition demanded relief. The first patient should probably have been operated on by the perineal route; in the third no general anæsthetic had at any time been possible.

Paroxysmal Tachycardia Treated with Quinidine.

DR. S. O. COWEN showed a male, *ætatis* seventy-five, who had been admitted to the out-patient department on November 29, 1923, complaining of attacks of palpitation. The attack started and stopped suddenly with a "jump" of the heart; they were more likely to occur after meals, but were likely to do so at any time without a recognizable cause. The first attack had occurred at the age of fourteen years. On two occasions they had been absent for nine months, but otherwise he had not been free from them for longer than two weeks. The duration of the attacks was from one-half to twelve hours, most of them lasting about an hour. On examination it had been found that the heart was somewhat enlarged, the peripheral vessels nodular and tortuous, the systolic blood pressure 155 and the diastolic pressure 110 millimetres of mercury. On several occasions he had been seen in an attack. The pulse rate was always 190 to 200 and after half an hour some cyanosis developed, but even in the longer attacks there was no other evidence of venous congestion. The offset of the attack was sudden, but it had been observed that it was preceded by a period of irregularity owing to extra systoles lasting several minutes. In the intervals extra systoles were frequent. Electro-cardiographic tracings showed that the ectopic focus from which the abnormal impulses arose, was situated in the auricle.

Three weeks previously the attacks had become more frequent, occurring every day and seldom lasting less than two hours. Examination had yielded the same information as that noted on his admission, except that the systolic blood pressure had altered to 170 and the diastolic to 90 millimetres of mercury. Quinidine sulphate in doses of 0·18 gramme (three grains) had been ordered three times a day and since its administration no further attacks had occurred, though the patient was still conscious of occasional extra systoles.

Scoliosis.

MR. C. J. O. BROWN, acting for Mr. Harold Dew, showed a patient, *ætatis* twenty-five, a labourer, who had first attended hospital on July 6, 1927, complaining of pain and limitation of movement in the region of the right hip. He had been sent from the country with a diagnosis of tuberculosis of the right hip. The pain was severe and shot down the outer side of the leg to the foot. He had also had night sweats and had lost some weight and had noticed that he was bent to the left side.

Four years previously he had had a similar attack of pain in the right hip which had lasted two weeks. Two years previously he had had pneumonia and had not felt well since. The family history was good.

On examination the patient manifested a gross lateral tilting of the whole trunk to the left side with no attempt to readjust the centre of balance. The lumbar lordosis was flattened and the right hip slightly abducted. The tilting of the trunk caused apparent protrusion of the right great trochanter. On palpation he was found to be tender over the point of exit of the right sciatic nerve from the pelvis, but not along the course of the nerve and the glutei muscles on the right side manifested myoclonic contractions on palpation. Movements of the right hip were full and free in all directions, but full flexion or extension caused slight tension pain in the gluteal region; similar movement of the left hip caused no pain. Movements of the lumbar part of the spine were limited and any movement caused pain in the gluteal region. Measurement showed that the limbs were equal in length and there was no tilting of the pelvis. X ray examination of the lumbar portion of the spine, sacro-iliac joints and right hip had revealed no bony abnormality. Rectal examination revealed no abnormality, the chest was normal. The temperature on two occasions had been 37·2° C. (99° F.).

The diagnosis made was sciatica with sciatic scoliosis and the treatment suggested was diathermy after a search for infective foci. The points of interest were:

1. The extreme grade of lateral bending without compensatory curves.

2. The story of night sweats and loss of weight suggest some infective condition.

3. Since movement caused pain the question of some form of support for the pelvis and lumbar spine deserved consideration.

DR. A. S. M. TYMMS said that he had never seen a case of sciatica with the same features as those described. The condition of the back was more important than the scoliosis and he suggested an X ray examination of areas higher up. There was certainly a limitation of movement in the spinal column and tenderness over the last dorsal vertebra so that it was quite possible that the lesion was higher up than the lumbar vertebræ.

MR. CHARLES OSBORN suggested a lesion around the sacro-iliac synchondrosis, as there was some tenderness there and it was not unreasonable to suggest that it was a sacro-iliac strain.

DR. S. O. COWEN said that the basis of the diagnosis of sciatica was that the maximum tenderness was over the sciatic nerve, that stretching of the nerve caused pain, that the ankle jerk was diminished on that side and there was 1·25 centimetres (half an inch) of wasting of that thigh. The position was unlike any sciatica that he had ever seen, yet he thought that the man must have a sciatic neuritis with a functional postural deformity added.

Rodent Ulcer Treated by Diathermy.

DR. W. KENT HUGHES showed a man, *ætatis* sixty, who six years previously had had a rodent ulcer on the left temple which had been present for six months. After one diathermy treatment there had been no recurrence. He stated that Wyeth was of the opinion that many cases of epithelioma and rodent ulcers were stimulated to growth by radiation.

Dr. Kent Hughes then demonstrated the use of the Wyeth diathermy knife.

Labyrinthine Investigation of a Case of Probable Cerebral Vascular Lesion.

DR. J. H. SHAW showed a patient of Dr. H. F. Maudsley, a male, *ætatis* forty-two, who had had a sudden onset of weakness on the right side four months previously. There had been no loss of consciousness and no complete hemiplegia. He had felt somewhat giddy at times and had also complained of tinnitus since the onset. He had noticed at the time that if he brought his right hand up to his face as in lighting a cigarette, his hand would tend to go out to the right, unless he watched it carefully. His condition had not altered since the onset.

Neurological examination showed the cranial nerves to be normal. The optic discs were normal. The deep reflexes on the right side were slightly more pronounced than on the left side, the right knee jerk inclined to be pendulous. The abdominal reflexes were normal, the plantars flexor. There was some past pointing to the right below the level of the shoulder which had only recently developed. With the finger-nose test the finger invariably went to the right. It was difficult to demonstrate any hypotonia on the right side.

THE NEWCASTLE HOSPITAL CLINICAL SOCIETY.

A MEETING OF THE NEWCASTLE HOSPITAL CLINICAL SOCIETY was held at the Newcastle Hospital on July 7, 1927. The meeting took the form of a series of demonstrations by the members of the honorary staff

Solitary Cyst of the Kidney.

DR. S. S. GARDINER showed a female patient who had been admitted to hospital suffering from attacks of left renal colic on and off for three months. Pyelograms had been taken, but nothing abnormal seen. The renal function had been within normal limits. The cyst had been easily enucleated from the kidney substance and the kidney sutured with catgut.

Dr. Gardiner said that in a recent article in the April, 1927, issue of *The British Journal of Surgery*, Fullerton had reported the ninety-ninth case recorded. He had stated that solitary cysts of the kidney were rare and that they were frequently caused by constriction of the tubules by sclerosis following chronic inflammatory changes. The cyst rarely communicated with the calyces or pelvis of the kidney and they were usually found on one side of the body only and at one of the poles of the kidney. They varied in size from that of a hazel nut to that of a man's head and were more frequently found in females. Symptoms might be entirely absent and the tumour in the abdomen accidentally discovered by the patient or the doctor.

The cyst described by Dr. Gardiner had been found on the convex border of the left kidney and measured 10 by 6·8 by 6·8 centimetres (four by two and three-quarter by two and three-quarter inches).

Trophic Ulcer of the Foot.

Mr. P., an ambulance officer, was presented suffering from trophic ulcer of the foot of two years' duration. He had been operated on for the correction of *pes cavus* and also had *spina bifida*.

As a result of discussion a new boot was being made and the ulcers exposed to the sun frequently during the day.

Dextrocardia.

DR. C. S. RENWICK, DR. C. A. MITCHELL AND DR. W. R. BEESTON joined in demonstrating two patients who were suffering from dextrocardia. They pointed out that dextrocardia was a condition in which the heart was placed in the right side of the chest cavity, instead of the left. It was usually associated with complete transposition of the other viscera of the thorax and abdomen.

Osler had stated that true dextrocardia was exceedingly rare as an isolated condition, but not very uncommon in combination with transposition of the other viscera.

Byron Bramwell had cited a case of congenital dextrocardia at Edinburgh in 1881 when no other organs were displaced.

Coates stated that often the organs were properly formed and their function was normal, although they are transposed.

Charles Creighton was of the opinion that the reversed position of the viscera was a developmental error depending on the retention of the right aortic arch as in birds, instead of the left as was usual in mammals. The position of all the unsymmetrical viscera was transposed, the spleen and cardiac end of the stomach going to the right side, the liver to the left, the caecum resting on the left iliac fossa and the sigmoid flexure of the colon being attached to the right. This condition of *situs inversus viscerum* need cause no inconvenience and it would probably remain undetected till occasion arose for physical examination or at a *post mortem* examination. In the two patients shown dextrocardia was associated with complete transposition of the viscera. In the second case the man was apparently moderately healthy, although his viscera were transposed. In the first patient there was a gross lesion of the heart valves and the boy was at times inconvenienced by his dextrocardiac condition. Probably he had been born with congenital deformity of the heart itself. A full account of dextrocardia was recorded in THE MEDICAL JOURNAL OF AUSTRALIA for September 4, 1915, by Professor Kellaway, of Adelaide.

Statistics showed that among 20,000 recruits examined for the war two manifested dextrocardia. At the Montreal and Royal Victoria Hospitals the condition had been noted twice in four thousand *post mortem* examinations.

Holt in his book on children's diseases stated that in 242 autopsies upon cases of congenital cardiac anomaly there were only two cases of dextrocardia. The Mayo clinic from 1910 to 1922 reported ten cases in 347,000 patients, so it could be concluded that the condition was moderately rare. In three years at the Royal Alexandra Hospital for Children Dr. Renwick had not seen a case and with the exception of a case seen at *post mortem* examination at the Royal Prince Alfred Hospital while he was a student he had seen no other cases but those demonstrated that night.

The pathology was obscure. Hirschfelder's theory was the generally accepted one, that in early embryonic life the position of the cardiac tube was changed by a reversal of the direction of the blood stream if the usual relation of the embryo to the primitive chorion was altered, so that the right instead of the left was placed nearer its blood supply.

The first patient shown was a male, aged ten years. The mother had always been delicate and had died of pneumonic influenza. The father was healthy and had been killed at the war. There had been no difficulty at the birth of the patient. He had apparently been healthy until five years previously, when he had a cold and on examination his dextrocardiac condition had been discovered. He had had several attacks of bronchitis since then. He had never had œdema, nor albumin in his urine. At times he had become breathless on exertion.

At the time of demonstration he was moderately well and attended school daily. On inspection an impulse was seen over the right side of the chest. The apex beat was in the nipple line on the right side in the fifth intercostal space. On palpation a distinct thrill was felt over the aortic area to the right of the sternum. On percussion the heart dulness extended from the second to the fifth rib on the right side of the chest. Laterally it extended from the right nipple line to 2·5 centimetres (one inch) to the left of the sternum. His liver dulness could be percussed on the left side. On auscultation there was a mitral regurgitant murmur conducted into the right axilla and again from the heart across the chest of the left axilla. A loud aortic regurgitant murmur was heard in the aortic area and extended through the aortic arch up into the vessels of the neck.

The condition present was congenital dextrocardia with complete transposition of the viscera, the heart giving signs of lesions of the aortic and mitral valves.

The second patient was a man, aged forty-two years, who had complained of epigastric discomfort, not amounting to pain, off and on for the previous six months.

This was as a rule relieved by taking food. No vomiting had occurred.

The patient had had enteric fever at the age of ten and attacks of "gastritis" eight years previously, but otherwise he had been healthy. He had played football as a schoolboy and had been a coal miner ever since. He had worked only part time of late owing to his stomach complaint. He had always been a total abstainer.

Examination revealed a complete transposition of viscera and X rays did not reveal any gastric or duodenal ulceration. The heart was apparently a normal organ, except for its position. The X ray films showed the position of the organs. In addition it was to be noted that the right testicle hung lower than the left and that there was a right-sided varicocele. The patient stated that he had always been conscious of the heart being on the right side.

X ray examination revealed complete transposition of the thoracic and abdominal viscera. The heart was more to the right side than the left, the apex beat being on the right. The aortic arch curved to the right instead of the left. No abnormality of the heart shadow, other than the dextrocardia, could be detected. The abdominal viscera were completely transposed. The cardiac orifice of the stomach was on the right side, the stomach was on the right and pointed to the left. The caecum was on the left side. The X ray films presented an absolutely normal appearance until it was noticed that the letters "R" and "L," denoting right and left sides, were transposed. No abnormality, other than the transposition, could be discovered.

Intracranial Tumour.

DR. I. MORGAN showed a male patient, aged seven years, who had been admitted to the wards of Newcastle Hospital on June 17, 1927, suffering from "abnormal movements of the right arm." The mother had stated that about two weeks prior to admission, the child was "out of sorts" and inclined to be drowsy. He had also complained of headache and had vomited on several occasions. One week prior to admission the mother had noticed some "twitching" movements of the right side of the face and had also noticed that the child was clumsy in attempting to perform movements with the right hand. He dropped his spoon and cup at meal times. His medical attendant, Dr. A. T. Roberts, who saw him at this time, had described choreiform movements of the right hand and arm. Within a few days the condition present on admission had developed.

On admission the child had been drowsy, had not complained of headache and had vomited occasionally. The cardio-vascular, alimentary and respiratory systems on examination had been found to be normal. The right arm had been held stiffly at the side and had resisted forcible movements. The wrist and fingers had been acutely flexed and the forearm had been held in an extreme position of pronation. The muscles of the upper arm and forearm had exhibited continuous clonic spasms, about one hundred per minute. During sleep the spasmodic contractions had disappeared. On admission the arm had become flaccid if the child's attention were distracted by some other strong stimulus. A few days after admission, however, the arm had remained contracted in spite of such stimuli. The right leg had shown similar contractions, but in a much less degree. Double papilloedema had been present when the child was admitted. A supranuclear paralysis of the seventh nerve had been present. No other cranial nerves had been involved. Paresis of the right leg had been definite and the knee jerk of this leg had been absent. The right leg had appeared to be hypotonic. The sensations of the right arm and leg had been normal. The left knee jerk had been present and active and both plantar reflexes had been flexor.

A lumbar puncture had been performed on June 22, 1927, and three cubic centimetres of fluid which was not under increased pressure, had been withdrawn. When examined, the cerebro-spinal fluid had been found to contain no abnormal constituents and had not reacted to the Wassermann test. On June 25, 1927, the left external rectus muscle had become paralysed. The facial paralysis had been more pronounced and the child had exhibited forced movements of rotation around the long axis of the body. At no time had there been an ataxia of a cerebellar type, nor did the child tend to fall towards one side.

On July 23, 1927, the swelling of the right optic disc had been estimated to be three diopters. The swelling of the left optic disc had been estimated to be one diopter. Nystagmus of the right eye had been recorded on June 30, 1927. The swelling of the optic discs had greatly increased and when examination was carried out on July 22, 1927, an upward deviation of the left eye had been noticed and a paralysis of the left external rectus was still present.

Commencing secondary optic atrophy had been noticed in the right eye and the swelling in both eyes had decreased somewhat. At no period during the illness had the temperature been elevated above normal and the respirations had remained constant at twenty-four per minute.

An X ray examination of the skull had not revealed any abnormality. The blood had been subjected to the Wassermann test and had not reacted. The blood had been examined on June 20, 1927, and the findings conformed to normal. At no time had there been any evidence of involvement of the eighth nerve.

In showing this patient Dr. Morgan stated that the absence of typical cerebellar ataxia and vertigo led him to believe that the lesion was not in the cerebellum itself. The presence of a supranuclear paralysis of the seventh nerve also suggested that the lesion was at the level of the seventh nerve nucleus or above it.

DR. J. BOSTOCK pointed out that the seventh nerve nucleus extended for a considerable distance in the *pons* below the point of emergence of the nerve itself. It was also pointed out, however, that if the nerve nucleus itself was involved, movements of the upper part of the face would not be present as they obviously were.

Dr. Morgan thought that the lesion involved the fibres of the facial nerve above the nucleus. The involvement of the sixth nerve might be a remote effect caused by increased intracranial pressure alone. It was difficult to explain the absence of knee jerks if the condition were a tumour of the cerebellum, as cerebellar tumours usually did not interfere with the mechanism controlling the reflexes such as the knee jerks, unless the intramuscular tension was transmitted to the spinal cord and involved the emerging nerves. Lumbar puncture, however, had shown that this was not the case. The tonic contractions of the forearm, the hypotonus of the leg and the forced movements of rotation all suggested that if the lesion itself were not in the cerebellum, then some of the cerebro-spinal tracts were involved. The picture very closely resembled the red nucleus syndrome described by Jeliffe and White.

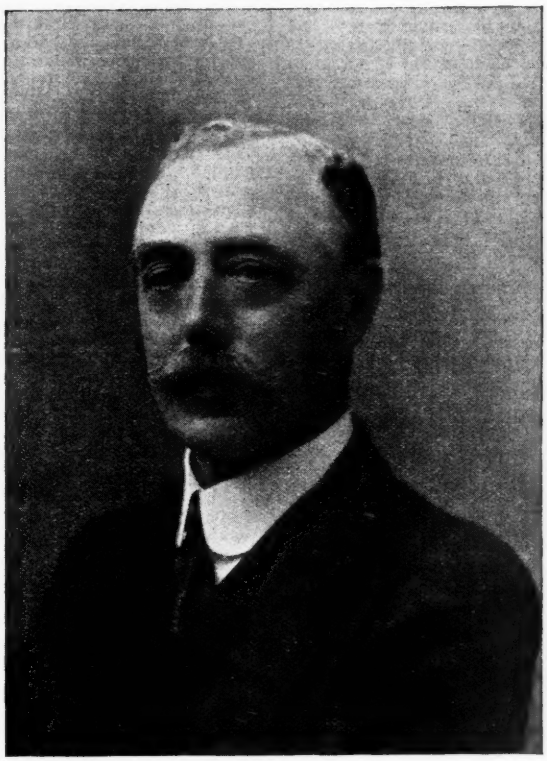
Mediastinal Tumour.

Dr. Morgan also showed a male patient, aged forty-four years, who had been admitted to the wards of Newcastle Hospital on June 24, 1927, complaining of pain in the throat for six months and cough of similar duration. He had also complained of periodical attacks of pain in the right side of the chest for four months. He had stated that the pain which he localized on the right side of the neck at the level of the hyoid bone, was of a lancinating character, appeared suddenly at any time and lasted for about one hour. He had complained also of dyspnœa on exertion and loss of weight, 6·3 kilograms (one stone) in six months. Some slight flattening of the chest wall over the upper lobe of the left lung had been noticed and it had been found on examination that the expiratory sounds were prolonged in this region. All other examinations had failed to reveal any abnormality.

On June 20, 1927, some bronchial breathing and a few sibilant rhonchi had been heard at the level of the third and fourth ribs on the right side about 6·25 centimetres (two and a half inches) from the edge of the sternum. A radiogram had been taken on May 16, 1927, and the radiographer had reported the presence of a well-defined patch of thickening, lying between the third and fifth ribs in front and occupying the middle portion of the right lung. The inner border had been about 1·25 centi-

metres (half an inch) from the tips of the transverse process and the outer border 1·25 centimetres from the lateral wall. The radiologist had been unable to detect any cystic appearance. He had made a diagnosis of either malignant disease or pneumonia. The patient had been discharged from hospital and had not been seen for three weeks prior to the meeting. An examination carried out that evening revealed definite dulness on percussion on the right side of the thorax posteriorly and a further X ray examination revealed by means of the fluorescent screen a large mass lying posteriorly to the mediastinum. The previous skiagram had been taken one month earlier and it was observed that a tremendous increase in the size and extent of the growth had taken place.

Members were inclined to think that the mass was a neoplasm growing in the posterior mediastinum. It was suggested that as the cough was not particularly severe and as there had been no hæmoptysis and very little sputum, the lung could not yet be involved to any great extent.



DR. W. R. BEESTON said that he had examined the patient with X rays that day and that there was an opaque mass, a little smaller than the clenched fist, occupying the middle third of the right lung. The central border was merged with the cardiac shadow and was of approximately the same density. The outer border extended to within about 3·75 centimetres (an inch and a half) of the lateral chest wall. The outline was irregular and not clear cut, as in the case of a cyst. The density was approximately the same throughout. On lateral examination the shadow was seen to lie behind the plane of the mediastinum and largely behind the plane of the vertebral column. The mass was rather flattened from before backwards. Its posterior border was about 2·5 centimetres (one inch) to 3·75 centimetres (one and a half inches) from the posterior chest wall. The heart was displaced slightly to the right. The mass did not pulsate. The mediastinum appeared to be fairly clear. The mass had increased considerably since the first examination made on May 3, 1927. The appearance of the mass suggested malignant disease.

Obituary.

ANDREW JOHN BRADY.

ON August 25, 1927, in the evening of a life spent for the benefit of humanity Andrew John Brady started on his last journey. Almost up to the last he had continued his professional and philanthropic work. While he was ill he had risen from his bed to attend a meeting of the Board of Directors of the Sydney Hospital of which he was President.

Andrew John Brady was born on July 23, 1849, at Swanlimbar in County Fermanagh, in the north of Ireland. His father and his grandfather before him had been landowners in those parts. He went to school at Pretoria in Enniskillen. In due course he left school and entered the medical school in Dublin, where he studied for the diplomas of the Royal College of Surgeons of Ireland and of the King's and Queen's College of Physicians of Ireland. In 1872 he secured the former and in the following year he obtained the diplomas of Licentiate of the King's and Queen's College of Physicians and Licentiate of Midwifery. A half of a century ago the medical course was different. Bacteriology had just been established; immunology as a science was non-existent; pathology was largely a matter of morbid anatomy; physiology was relatively crude and elementary as compared with today. But the physicians of then were wise men, wonderful observers and shrewd diagnosticians. Brady made the most of the practical training of his day and emerged from the school with a sound grounding of professional knowledge. His uncle, the late John Moffat, was in medical practice in Sydney and it was arranged that he should travel half round the world to start his remarkable career. His uncle and he conducted a general practice from a house in Castlereagh Street, where the City Mutual Life Insurance Company now has its offices. He was appointed to a resident position at the Sydney Hospital shortly after his arrival in Australia. A little later he moved to Lyons Terrace, where he resided and practised until 1911. From the first he took an active interest in the affairs of his profession and was recognized by his colleagues as an unusually strong and resourceful partisan. In 1880 the Sydney and New South Wales Branch of the British Medical Association came into being and obtained recognition by the Central Council. Two years later Brady was appointed a member of the Council of the Branch. His keenness, his equitable mind, his sound judgement rendered him a particularly valuable councillor. He was re-elected once and then withdrew from nomination because he determined to visit Europe for a special purpose. For over a year he worked at the celebrated ear, nose and throat clinic in Vienna under the great Politzer. This special training laid the foundation for his future activities, but it did much more. Up to that time there were no throat specialists in Australia. Andrew John Brady recognized that Australian medicine suffered on this account and he made it his business to set a high standard for others to emulate. On his return he continued his special studies and it was at once apparent that this young man would soon be known all over the world as a leader and as an authority. Two years before he visited Vienna he was appointed Honorary Surgeon to the Sydney Hospital and was elected the representative of the honorary medical staff on the Board of Directors. In 1884 he acted as Honorary Treasurer of the New South Wales Branch of the British Medical Association. After his return he worked steadfastly for the institution of a special department for diseases of the ear, nose and throat at the Sydney Hospital. His colleagues and his associates on the Board of Directors were so impressed by his energy and argument that the depart-

ment was created in 1887 and placed under his care. The soundness of this innovation was immediately apparent. Not only was his clinic crowded with patients who derived great benefit from Brady's skill and knowledge, but many of the young graduates attended to learn what he had to teach concerning the commoner and rarer diseases of the ear, nose and throat. The authorities of the Royal Prince Alfred Hospital feared that they would suffer by comparison if a similar department were not established at their hospital. Andrew John Brady was invited to become specialist in diseases of the ear, nose and throat at the Royal Prince Alfred Hospital and he accepted the invitation.

At this time Brady recognized that the pharynx, fauces and larynx were in intimate pathological relationship with the teeth and that his work at the hospitals was being restricted and hampered because facilities for dental treatment were not provided. He therefore persuaded the Directors of the Sydney Hospital to institute a dental department; it proved of immense benefit to the patients. In addition his experience in England and on the Continent had taught him that the physician and surgeon depended to a large extent for success on skilled nursing. At that time there was no training school for nurses and he pressed for this instrument to raise the standard of nursing in Australia. It need scarcely be emphasized in the year 1927 how important this movement was. While he was working quietly but with the expenditure of much energy at his own specialty, his prescience led him to be the prime mover in the establishment of many small and large reforms in medical work. In 1886 he married Miss Campbell, a lady who aided him in adding to his achievements, while she herself, infected by his urge to do things, has been actively concerned in innumerable public and semipublic activities.

In 1893 Brady was again elected a member of the Council of the New South Wales Branch of the British Medical Association. In 1894 he again visited Vienna and he also spent time in London, studying the methods of the eminent throat surgeons of the day. His own methods were sound and some were original, but he was always anxious to learn from others and to adopt procedures which appealed to him to be good. In the years that followed his return he manifested the same spirit of reform and initiation, the same keenness to lead and to benefit humanity as of yore. In 1899 he was prevailed on to stand for election to the Council of the New South Wales Branch of the British Medical Association. From that date until he asked his colleagues not to insist on his service in 1923, he was elected year after year. In 1902-1903 he became Vice-President of the Branch and in the following financial year he occupied the President's chair. Not only did he take a prominent and active part in the medico-political activities of the Branch; he also was keenly interested in the various sessions of the Australasian Medical Congress. He contributed occasionally to the literature of his specialty and his articles were always instructive and important. Several of his best articles appeared in the *Journal of Laryngology*, including one descriptive of his operation for the treatment of fibromata of the naso-pharynx. Within recent years his pen has not been busy and only a few articles have been given to the medical profession of Australia through this journal.

In the early years of the war Andrew John Brady, together with the late George E. Rennie and Professor D. A. Welsh, formed a military medical board. He rendered sterling service for his country in this way. He held a commission in the Reserve with the rank of Major. His opinion was sought and acted on by the Commonwealth Government in matters of national hygienic importance.

The secret of his success was not far to seek. Apart from his skill and knowledge and from his unswerving determination to achieve his objectives, there is the fact that Andrew John Brady was a courtly gentleman, lovable, sensitive to the sufferings of humanity, gentle and large hearted. His reputation was world-wide. In Australia he was respected by all and revered by those with whom he came in intimate contact. He gained the complete confidence of the lay members of the Board of Directors of the Sydney Hospital. This institution is indebted to him for more than the mere service over a period of fifty-three years. He did not appear to grow old. He was possessed of excellent health until an attack of influenza with fever and digestive symptoms occurred. This was during the week ended August 20, 1927. Later acute peritonitis supervened and ran a rapid course.

Mention should be made of Brady's association with the Rod Fishers' Society of which body he became President. He was a first class pisciculturist and was associated with the keenest fishermen of Sydney. He was also a good golf player. He is survived by Mrs. Brady, two daughters, Mrs. Ernest Watt and Miss Moira Brady, and one son, Mr. Gordon Brady, D.S.O., M.C., of whom he was very proud. The loss to the medical profession is very great, despite the fact that he had passed the proverbial span of three score and ten. His death is a serious loss to the community. The heart-felt sympathy of the medical profession goes out to his widow and her family.

DR. RICHARD ARTHUR writes:

I was Dr. Brady's assistant at the Sydney Hospital for a good many years. This has always remained to me a pleasant memory, for I found him the very soul of courtesy and consideration. And whether he had an innate sunniness of temperament or had himself marvellously under control, I never saw him anything but calm and serene.

He suffered the foolish with an infinite patience and seemed devoid of any trace of professional envy or jealousy. Perhaps this was because he felt himself, as he was rightly entitled to do, so easily first in his branch of the profession. He was always glad, however, to place what knowledge he had at the disposal of his colleagues; that knowledge was wide and comprehensive. And though

he gave the impression of being slow and deliberate in his thoughts and movements, he always arrived.

As a trivial instance, I look back on a mistaken prophecy I once made when I saw him beginning to learn golf; I said that was one thing he would never be any good at. I understand that later he became a very fair player. He will be greatly missed at the Sydney Hospital Board, where he presided with the same courtesy and equanimity he showed elsewhere and all of us who knew and admired him, will feel that a very perfect gentleman has gone from our midst.

GEORGE McINTYRE MAGGS.

DR. GEORGE MCINTYRE MAGGS, whose death was recently reported, was born on May 21, 1901. He received his early education at the State School. He entered Scotch College, Melbourne, in 1914 and two years later passed the Junior Public Examination with two distinctions. In 1918 he passed the school leaving examination and in the following year entered the University of Melbourne and commenced the study of medicine. He graduated in August, 1925, and in the following month became a member of the British Medical Association. For a short time after graduation he did *locum tenens* and insurance work and then obtained the position of Resident Medical Officer at the Austin Hospital and later at the Infectious Diseases Hospital, Fairfield. At the latter hospital he developed during the course of his duties a small pustule on the finger which was followed by septicæmia. Two days before his death he received notification that he had been appointed Resident Medical Officer at the Brisbane Hospital. He was keenly interested in all branches of sport and at Scotch College he rowed in the second eight. He was very fond of music and was the life of the "Medical Medleys," a musical society which gave several performances in aid of charity. His bright and cheerful disposition, his willingness to lend a helping hand and his kindly sympathy to those around him made him deservedly popular wherever he went. He took a very keen interest in his profession and his death just at the beginning or his career was deeply felt by a large circle of friends. The medical profession offers sympathy to his father and mother.

Dr. Ian McInnes writes:

His charming personality and sympathetic manner endeared him to all who crossed his path, and his genial *camaraderie* and good nature assured him of a popularity that was tremendous amongst his fellow students. He was inherently unselfish and was never heard to say an unkind or mean thing of any fellow being. As their only son his parents must find their loss irreparable and his untimely death at the very threshold of his career will leave a gap that cannot be filled in the hearts of those who were honoured by his friendship.

Hospitals.

THE CANCER WARD OF THE BENDIGO BENEVOLENT ASYLUM.

DR. O. PENFOLD, the medical officer in charge of the Cancer Ward of the Bendigo Benevolent Asylum, has issued a report dated August 8, 1927. This report shows that useful work is being done at that institution in alleviating the sufferings of patients in the last stages of malignant disease. The various hospitals in Melbourne had become overcrowded with patients and the Charities Board, in pursuance of its policy of decentralization, requested the Committee of the Bendigo Benevolent Asylum to take charge of male patients with inoperable malignant disease. Accordingly, in July, 1925, a ward was opened, equipped and staffed for the reception of twenty patients. The whole of the expense was borne by the Charities Board. From the time of its opening until July 31, 1927, thirty patients have occupied the beds. Twenty-two patients have died, the high mortality rate being due to the advanced stage of the disease in all cases. Two patients have left the institution, one to go to friends in Melbourne and the other to try to do some light work in Bendigo. On July 31, 1927, six patients remained in the ward. The Charities Board has been so gratified with the manner in which the work has been carried out, that it has requested the Committee to prepare a similar ward for the treatment of women suffering from cancer. This has been done and the ward is now nearly ready for the reception of twenty patients.

The patients who are admitted to the ward are as a rule without means and have neither relatives to support them nor friends to take any personal interest in them. In these circumstances and because of the hopeless condition of the sufferers from the point of view of cure, it will be seen that the Cancer Ward already forms an important link in the hospital activities of the State of Victoria. Dr. Penfold points out that in addition to treatment aimed at the alleviation of suffering, measures are undertaken to bring as much brightness as possible into the lives of the inmates.

The average age of the thirty patients already mentioned was 67·5 years. The oldest was ninety-eight and the youngest forty-seven years of age. The average age at death of the twenty-two who died was 67·68 years. The parts of the body affected in the thirty patients were as follow: Lip, five cases; face, three; tongue, four; head, two; mouth, four; jaw, two; bowel, five; stomach, two; prostate, two; heel, one. The average stay in the ward of each patient was eighteen and a half weeks. The longest period for any one patient was sixty-eight weeks and the shortest period one week.

University Intelligence.

THE UNIVERSITY OF SYDNEY.

A MEETING OF THE SENATE OF THE UNIVERSITY OF SYDNEY was held on September 5, 1927.

The Senate has been advised by the Secretary of the British Empire Cancer Campaign that the Grand Council has granted affiliation to the British Campaign to the Cancer Research Committee of the University of Sydney.

Dr. A. Y. Fullerton presented to the University an X ray tube for the Department of Physics and a small library of medical publications.

The collection of *Amycterides* of the late Dr. E. W. Ferguson has been presented by Mrs. Ferguson to the Macleay Museum of the University.

The Council for Scientific and Industrial Research has made a grant of £250 to the University for the purpose of radio investigations.

Miss E. A. Stobo was appointed a Demonstrator in Chemistry from January 1, 1928.

Proceedings of the Australian Medical Boards.

VICTORIA.

THE undermentioned has been registered under the provisions of Part I of the *Medical Act*, 1915, of Victoria, as a duly qualified medical practitioner:

Allan, Robert Marshall, M.D., 1914, F.R.C.S., 1919 (Edinburgh), c.o. Victorian Branch, British Medical Association, Albert Street, East Melbourne.

Additional diploma registered:

Francis John Niall, M.D. (Melbourne), 1927.

Books Received.

THE METHODS OF CLINICAL DIAGNOSIS, by Alexander George Gibson, M.D., F.R.C.P., and William Tregonwell Collier, M.D., M.R.C.P.; 1927. London: Edward Arnold and Company. Crown 8vo., pp. 406, with illustrations. Price: 12s. 6d. net.

Diary for the Month.

SEPT. 19.—New South Wales Branch, B.M.A.: Organization and Science Committee.
SEPT. 20.—Tasmanian Branch, B.M.A.: Council.
SEPT. 20.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
SEPT. 21.—Western Australian Branch, B.M.A.: Branch.
SEPT. 21.—Section of Obstetrics and Gynæcology, New South Wales Branch, B.M.A.
SEPT. 23.—Queensland Branch, B.M.A.: Council.
SEPT. 23.—South Eastern Medical Association (Wollongong), New South Wales.
SEPT. 23.—Illawarra Suburbs Medical Association, New South Wales.
SEPT. 24.—Eastern District Medical Association (Taree), New South Wales.
SEPT. 27.—New South Wales Branch, B.M.A.: Medical Politics Committee.

Medical Appointments.

Dr. Emanuel Sydney Morris (B.M.A.) has been appointed an Acting Member of the Aborigines Protection Board, New South Wales.

* * * *

Dr. Charles George McDonald (B.M.A.) has been appointed an Acting Member of the Board of Official Visitors to the Cook's River Mental Hospital, Sydney.

* * * *

Dr. Charles Nathaniel Simons (B.M.A.) has been appointed Government Medical Officer at Tweed Heads, New South Wales.

* * * *

Dr. David Aiken (B.M.A.) has been appointed Government Medical Officer at Murwillumbah, New South Wales.

* * * *

Dr. W. B. Fry (B.M.A.) has been appointed District Medical Officer and Public Vaccinator at Lake Grace, Western Australia.

* * * *

Dr. Errol Solomon Meyers (B.M.A.) has been appointed a Licensed Teacher of Anatomy, School of Anatomy, University of Queensland.

* * * *

Dr. James Sydney Alexander Rogers (B.M.A.) has been appointed Acting Medical Superintendent of the Hospital for the Insane, Beechworth, Victoria.

* * * *

Dr. David Duncan Cade (B.M.A.) has been appointed Acting Medical Superintendent of the Hospital for the Insane, Mont Park, Victoria.

* * * *

Dr. Stanley Argyle (B.M.A.) has been appointed a Member of the Council of the University of Melbourne, Victoria.

Medical Appointments Vacant, etc.

For announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser," page xviii..

INFECTIOUS DISEASES HOSPITAL, FAIRFIELD, VICTORIA: Junior Resident Medical Officer (Male).

LAUNCESTON PUBLIC HOSPITAL: Junior Resident Medical Officer (Male).

MELBOURNE HOSPITAL: Assistant Anæsthetists (2), Medical Clinical Assistants (2), Surgical Clinical Assistants (2).

Medical Appointments: Important Notice.

MEDICAL practitioners are requested not to apply for any appointment referred to in the following table, without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCH.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 30 - 34, Elizabeth Street, Sydney.	Australian Natives' Association. Ashfield and District Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Petersham Dispensary. Manchester United Oddfellows' Medical Institute, Elizabeth Street, Sydney. Marrickville United Friendly Societies' Dispensary. North Sydney United Friendly Societies. People's Prudential Benefit Society. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
QUEENSLAND: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane.	Members accepting appointments as medical officers of country hospitals in Queensland are advised to submit a copy of their agreement to the Council before signing. Brisbane United Friendly Society Institute. Stannary Hills Hospital.
SOUTH AUSTRALIAN: Secretary, 207, North Terrace, Adelaide.	All Contract Practice Appointments in South Australia. Booleroo Centre Medical Club.
WESTERN AUSTRALIAN: Honorary Secretary, 65, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.
NEW ZEALAND (WELLINGTON DIVISION): Honorary Secretary, Wellington.	Friendly Society Lodges, Wellington, New Zealand.

Medical practitioners are requested not to apply for appointments to positions at the Hobart General Hospital, Tasmania, without first having communicated with the Editor of THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, New South Wales.

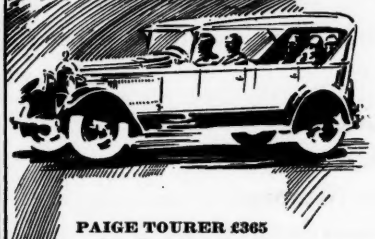
Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

All communications should be addressed to "The Editor," THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, Sydney. (Telephones: MW 2651-2.)

SUBSCRIPTION RATES.—Medical students and others not receiving THE MEDICAL JOURNAL OF AUSTRALIA in virtue of membership of the Branches of the British Medical Association in the Commonwealth can become subscribers to the journal by applying to the Manager or through the usual agents and booksellers. Subscriptions can commence at the beginning of any quarter and are renewable on December 31. The rates are **£2 for Australia and £2 5s. abroad *per annum* payable in advance.**

PAIGE 4-DOOR SEDAN, £525.

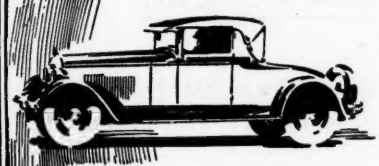


PAIGE TOURER £365
The Touring Car
De Luxe.

PAIGE

"The Most Beautiful Car in Australia."

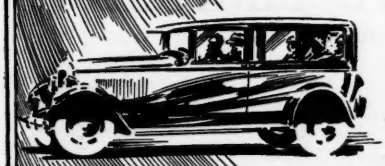
Today the style leadership of the Paige is unquestioned. Paige bodies are beautifully moulded and finished, and beneath this beauty are chassis, by all tests, the finest Paige has ever built. For nearly twenty years Paige cars have been noted for dependability and unfailing reliability.



CABRIOLET ROADSTER
£545.
For mechanical perfection and completeness of appointments this model is supreme.

Paige cars are as honestly priced as they are honestly built. You cannot buy a quality motor car for less than the price of a Paige. You will always regret it if you don't buy one of these beautiful cars. Fully equipped, nothing extra to buy.

Very Easy Terms by Arrangement.



PAIGE STRAIGHT EIGHT £875.
With Hi-Flex Transmission and a host of other improvements.

Marquette Motors Ltd.

Paige Showrooms and Service Station:
117-31 Flinders Street, Moore Park
(Near Anzac Parade).
Authorized Dealers:
PAIGE MOTORS, 197
Metropolitan Motors, 163

For Nervous Irritability

TRADE MARK **'LUMINAL'** BRAND

PHENOBARBITAL

TABLETS

As a sedative and antispasmodic in nervous irritability "Luminal" is constantly gaining in favour. It acts efficiently in much smaller doses (¼ to ½ gr.) than are required in epilepsy.

Among the conditions in which especially good results have been noted are the following:—

Chorea.	**Pertussis.**
Neurasthenia.	**Exophthalmic Goitre.**
Cardiac Neuroses.	**Gastric Neuroses.**
Climacteric Disorders.	**Dysmenorrhœa.**

Pre- and Post-Operative Cases.

℞ **"Luminal" Tablets ¼ gr. Original bottle of 100.**

"Luminal" Tablets are also supplied in 1-gr.-bottles of 50; 1½-gr.-tubes of 10, and bottles of 50 and 100; 3-gr.-tubes of 10; 5-gr.-tubes of 10.

Therapeutic data and Clinical Observations post free to the Medical Profession, on request.

BAYER PRODUCTS LIMITED

19, St. Dunstan's Hill, London, E.C.3.

SOLE CONSIGNEES:

—Fassett and Johnson, Ltd., 233, Clarence Street, Sydney.

—Fassett and Johnson, Ltd., P.O. Box 33, Wellington.

Seamer Street, Glebe, Sydney, N.S.W., by HENRY WILLIAM ARMIT, AUSTRALASIAN MEDICAL PUBLISHING COMPANY, LIMITED, h Street, Sydney.